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OF
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AND
LARYNGOLOGY
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VOL. XLI. DECEMBER, 1932. No. 4.

LXXVII.

REPORT OF A CASE OF PAPILLARY CARCINOMA OF THE MAXILLARY SINUS.*

JOSEPH HAMMOND BRYAN, M. D.,

WASHINGTON, D. C.

In presenting the report of this variety of carcinoma of the maxillary sinus, it is with the view of placing it on record, showing the beneficial effects of radium on malignant growths. Of course, it may be said it is too soon to claim any permanent results from radium—only a little over six months has passed since the patient was operated upon. With that end in view, I am presenting this as a preliminary report of a case of malignant disease of the maxillary sinus treated with radium. If in the future any return of the growth is noted a report will be made.

Miss C., aged 36, came to my observation October 3, 1931, complaining of severe pain over the left maxillary sinus, the pains radiating to the frontal region, the lower jaw and the left ear. Family history negative.

She gave the following personal history: During the early part of last winter she contracted influenza, resulting in an abscess

*Read before the Fifty-fourth Annual Congress American Laryngological Association, Atlantic City, May, 1932.

in the left ear and excessive secretion from the left side of the nose. A myringotomy was done, and in due course the abscess in the ear healed. The left antrum was punctured frequently for a supposed abscess in the left maxillary sinus by her former medical attendant. These punctures were made four or five times a week in the early stage of her illness. The punctures were very painful and not followed by much secretion or relief from the pain in the face. There was always a severe hemorrhage following each puncture, which persisted in the intervals of the punctures. She also complained of occlusion of the left nostril, which caused her great inconvenience, made her very nervous, and annoyed her greatly while at work as typist.

Examination showed a young woman, well nourished but extremely nervous. There was some swelling over the left antrum and great pain palpating over this sinus. The left nostril was completely occluded; the right side remained open, through which she was able to breathe. As far as could be determined at this examination, there was no evidence of a growth in the nostril. The heart and lungs were apparently normal. A further examination revealed she had a large cauliflower growth on the dorsum of the left foot, very vascular and bleeding readily. She had been under a number of dermatologists, who diagnosed this growth as a naevus, and had frequent exposures to the X-ray without any diminution in the size of this growth. She stated that while dancing one evening her partner stepped on her foot and a profuse hemorrhage resulted, necessitating her removal to a hospital for treatment.

A report of X-ray examinations of the nasal accessory sinuses by Drs. Groover and Christie shows marked opacity over the left antrum and slight cloudiness of the corresponding ethmoid cells. The other sinuses are practically clear. There appears to be considerable deformity of the nasal septum with occlusion of the left nostril.

The following is a report of Dr. Louis S. Greene, ophthalmologist, to whom she was referred for an examination of her eyes: Vision in both eyes 20/20; the fields for colors and white are normal save they are of the fatigue type, more marked in the left eye than in the right; the blind spot in the left eye is markedly

enlarged. There is no exophthalmus. The ophthalmoscope shows a moderate degree of congestion of the retinal vessels in the right eye, while those in the left eye are not only congested but tortuous, and there is a slight retinal edema. There is a left hypophoria of between ½ to 1 degree, and pain in the left eye is elicited by rotating the eyes upward. The above presents a picture of both toxic and mechanical interference of the left eye, and, judging from past experience, I would say that the antrum on the left side was a decided contributing factor. The whole examination presents a picture of disturbance in the accessory sinuses on the left side.

The patient was sent to the Episcopal Eye, Ear and Throat Hospital, and on October 8th, I did a radical operation on the left maxillary sinus. On opening the cavity it was found filled with a large papillomatous growth which was very vascular. The removal of the growth was attended by a very profuse hemorrhage, which was with great difficulty checked. After the removal of the growth the nasal wall and the posterior wall of the cavity were found to be markedly necrotic. The cavity was packed with iodoform gauze and then the patient was returned to her room in very bad condition. I was hurriedly sent for and found the patient in a state of collapse. Administered ten minims of adrenalin hypodermically, and as soon as the blood from a donor could be obtained 100 c.c. of blood was injected intravenously. She rallied slowly, and in about two hours her color returned and her pulse became stronger, about 124. The Murphy drip was ordered and continued for three days. After that time the salt solution could not be retained rectally by the patient and it was discontinued.

Report of the examination of the growth by the pathologist, Dr. Selinger: Section of the growth shows groups of large, irregularly shaped atypical epithelial cells of varying sizes. There is a definite tendency towards a papillomatous arrangement. Besides groups above mentioned, these cells are seen scattered irregularly throughout the tissue. Mitotic figures are frequent. Diagnosis: Carcinoma, papillary type.

The wound was dressed on the second day after the operation and the cavity irrigated with normal salt solution. The removal of the gauze was attended with severe pain and profuse bleeding.

Fig. 1. Shows low power magnification atypical area, epithelial cells.

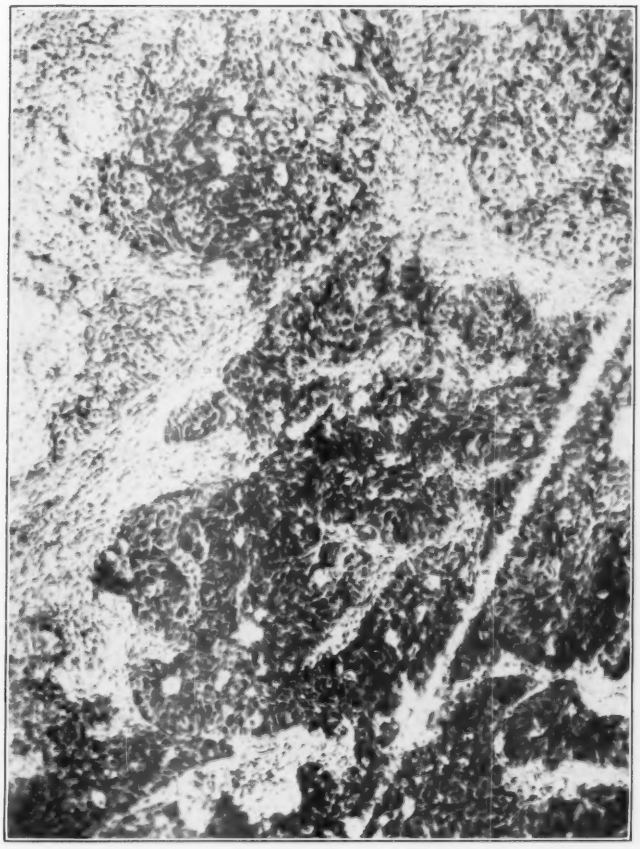

Fig. 2. Shows high power magnification. Mitotic figures frequent.

The cavity was packed with gauze dressing, which was allowed to remain forty-eight hours. The removal of the gauze was attended with such excruciating pain that the gauze dressings were abandoned and a gutta percha obturator was placed in the canine fossa to keep the wound from closing. The cavity was irrigated every second day, the secretions diminishing rapidly and the interior of the sinus looking well.

October 19th. The patient had progressed so rapidly she was allowed to go to the office of Drs. Groover and Christie, who applied the X-ray to the left cheek 20 x 200kv x 10/60 x 60½ m. cu. and 1 mil. al.

October 21st. She has had great pain in the back of the head since last X-ray treatment. Face feels drawn and swollen. The left cheek was treated again with X-ray 20 x 200kv x 10/60 x 60½ m. cu. and 1 mil al.

October 22nd. Fifty mg. of radium in platinum needles and brass rubber covered capsules were inserted in the left antrum to remain 68 hours. The capsule was packed away from the antral wall as well as possible. It came out that evening about 10:30, and passed backward into the nasopharynx. It was removed by the house surgeon; she came near swallowing the capsule. The time the capsule was in the antrum equaled about 375 mg. hours.

October 27th. Fifty mg. radium were reinserted in the left antrum and anchored by a strong linen string passed around the neck.

October 29th. Eight p. m., the radium was removed; equals 2,850 mg. hours.

October 30th. The patient complains of intense sore throat, swelling and redness of the fauces were noted. An antiseptic mouth wash and crushed ice by mouth was ordered.

November 2nd. Marked swelling of the cervical glands on the left side, very painful, fauces badly swollen, and red, white, patchy condition of the roof of the mouth and dorsum of the tongue. A culture taken from the fauces and postnasal space showed streptococcus viridans. The radium reaction was intense and deglutition was very painful.

November 30th. Patient complains of pain in the jaw on the left side. She looks well and the reaction from the radium has completely subsided. No discharge from the sinus. As the obturator was causing her discomfort, owing to the closure of the wound in the anterior wall, and the interior of the cavity looking well, and no evidence of any return of the growth, the obturator was removed.

December 17th. The patient looks well; nose, pharynx and mouth look normal. Up to the present writing there is no evidence of a return of the growth, and otherwise in good condition except for a highly nervous condition. The patient was allowed to take a sea trip to the West Indies.

April 25th. She has just returned from her trip to the West Indies looking the picture of health and having lost all of her nervous symptoms. The antrum was irrigated with salt solution through a small opening remaining in the canine fossa, and return flow shows no secretion present; normal respiration through left naris, and she is free from pain over the antrum and fauces.

She was discharged, to report occasionally for observation. Owing to the intensive work that is being done on cancer all over the world, I believe all cases treated with X-ray and radium should be recorded.

302 Stoneleigh Court.

LXXIII.

HISTOPATHOLOGY OF THE LARYNX ANTEDATING CLINICAL EVIDENCE OF LARYNGEAL TUBERCULOSIS.*

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BOULDER, COLO.,

AND

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PHILADELPHIA.

Recognizing the well known fact that tuberculous infections in the epiglottis, base of the tongue and pharynx produce dysphagia and odynphagia sooner, and in more severe form than tuberculous lesions in the interior of the larynx, and believing that many patients suffering with pulmonary tuberculosis, and with sputum laden with tubercle bacilli, must have early microscopic tubercles in the larynx long before gross lesions are clinically demonstrable, we undertook the gross and microscopic study of the larynx, epiglottis, base of the tongue and pharynx at postmortems using autopsy specimens of patients who had pulmonary tuberculosis and in whom pulmonary tuberculosis was the chief cause of death. We purposely did not use specimens from patients with demonstrable gross laryngeal tuberculosis before death, except in two of the cases. These two had only incipient lesions. Wherever possible, negative and unimportant examinations, tests, etc., have purposely been omitted from the paper.

Each larynx was competely removed at the autopsy with the tongue and pharynx attached. These organs were carefully examined for gross lesions, but none were found in four of the six

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*Read at the meeting of the American Laryngological Association, Atlantic City, N. J., May 19, 20 and 21, 1932.

From the Departments of Otolaryngology and Pathology, University of Colorado School of Medicine and Hospitals, and the National Jewish Hospital for Consumptives, Denver, Colorado.

specimens used for this study. Small pieces of tissue were cut from the larynx, epiglottis, base of the tongue and pharynx for microscopic sectioning.

The abstracts of the case histories are as follows:

Autopsy 827.—Mrs. S., age 33, white, American; occupation, nurse. Mother, father and sister died of tuberculosis. Admitted to the National Jewish Hospital for Consumptives, May 5, 1929; died April 5, 1930. There were no laryngeal symptoms until four weeks prior to death. The principal complaint was dryness and slight difficulty in swallowing. All laryngeal examinations were negative. At admission she gave a history of pulmonary tuberculosis for nine years. During this entire time the course of the disease was progressive. The physical examination revealed the right side completely excavated with some slight findings at the apex posteriorly. During her hospital residence she had two moderate sized hemorrhages, from which she recovered without complications. Up to September, 1929, she ran a septic temperature, generally as high as 102° F., every afternoon, but felt remarkably well in spite of this. The pulse was 120 at rest.

On September 12, 1929, a phrenectomy was performed on the right side. There was an improvement in the temperature afterwards, so that it was below 100° F. during the remainder of her residence. On several occasions she developed edema of the ankles and eyelids, which cleared up under ordinary treatment. The blood chemistry and urine analyses were negative until several days before death. On March 19, 1930, she developed severe intestinal symptoms, severe cramps and diarrhea with increased edema of the ankles and face. The urine at this time showed large quantities of albumin and casts. This was considered a terminal tuberculous enterocolitis with possible amyloid degeneration of the kidneys.

The sputum in this patient was persistently positive. The sedimentation rate was 128, and it did not go below this during her entire residence. The roentgen-rays at the time of her admission showed complete opacity over the entire right lung with some density over the left apex and some scattered fibrous and inflammatory mottling below this. The heart and mediastinum were displaced to the right. Another plate, taken in September,

1929, showed no changes from the former examination. The patient died April 5, 1930.

Anatomic Diagnosis.—Pulmonary tuberculosis with cavitation, bilateral. Tuberculous colitis with ulceration. Amyloid infiltration in spleen, adrenals and kidneys. Fibrous adhesions of pleura, bilateral. Edema of lower extremities. Healed phrenectomy scar on right side of neck. The right pleural cavity was obliterated and the left had adhesions over the apex and base. The right lung was completely riddled with cavities. On the left side the upper lobe was well aerated while the base was firm. There were many caseous nodules, especially about the left hilus. The cecum had numerous ulcerations. The spleen was greatly enlarged. The bladder had congestion and fine hemorrhages in the trigone and over the posterior wall.

The epiglottis appeared normal. The arytenoids appeared slightly soft and thickened. The rest of the larynx and the vocal cords appeared normal.

Microscopic Examination.—The lungs had cavities lined by or filled with polymorphonuclear leucocytes. There was considerable caseation in large areas, with giant cells, and surrounded by fibrosis, outside of which the alveoli were compressed and atelectatic. There were ulcers in the large intestine. The spleen had a large amount of amyloid replacing the malpighian corpuscles and extending beyond these. The adrenals and the kidneys showed considerable amyloid. The base of the tongue mucosa was intact and appeared normal. The stroma was dense and had a few lymphocytes. The epiglottis had a small loss of epithelium from the tip, in the vicinity of which the stroma was slightly edematous and had a few large mononuclear cells, polymorphonuclear cells and lymphocytes. The lower part had fairly compact stroma. The laryngeal epithelium was intact. The stroma was fairly dense and thin.

Comment.—There was no gross clinical evidence of laryngeal tuberculosis in this patient before death and no histopathology of tuberculosis in the larynx after death.

Autopsy 754.—Mrs. D. was admitted to the University of Colorado General Hospital on October 1, 1929. Her chief complaint

was of chronic productive cough, night sweats, afternoon fever and loss of weight.

The patient was first admitted to Colorado Psychopathic Hospital, on the campus at the Medical School, September 13, 1929. She had attacks beginning with a gripping pain in the right side, followed in a day or so by passage of much mucus and blood. She had suffered since the age of seven. She was never constipated. The last attack occurred July 3, 1929, and lasted six to seven weeks. She lost about twenty-five pounds. She was bedfast most of the time. The stools at first contained mucus and blood. Later they were greenish with a bad odor. She had considerable nausea and vomiting. After entering the psychopathic hospital she had night sweats, cough with sputum and afternoon fever. There were no throat symptoms.

She had smallpox, mumps and influenza in 1928. She was sick six to seven weeks with influenza and expectorated blood.

Physical Examination.—The tonsils were hypertrophic. The pharyngeal mucosa was hyperplastic. The nose and larynx were negative. The right lung was hyperresonant. There were no râles. The right apex was clear. The left lower lobe was resonant. There were no râles. The left apex was dull with loud crackling râles, anteriorly and posteriorly. In an area over the fourth interspace in the anterior axillary line, amphoric breath sounds were heard. The voice sounds were increased over this area.

Clinical Diagnosis.—Pulmonary tuberculosis with cavity formation.

Summary.—The patient was transferred from the Colorado Psychopathic Hospital to the Colorado General Hospital with extensive pulmonary tuberculosis. On the fifth day pneumothorax was attempted unsuccessfully. On about the twelfth day she had severe headache, followed by slight rigidity of the neck. Kernig's sign was positive. There was nysgtamus and the pupils were unequal. One spinal puncture showed high protein and many lymphocytes, but no tubercle bacilli. Cisternal puncture three days later showed increased pressure, many lymphocytes and acid-fast bacilli. The patient died October 26, 1929.

Laboratory Report.—Urine: Sp. G., 1,010 to 1,028. Occasional hyaline casts. Pus cells 1 to 2; epithelial cells 1 to 2. Occasional

red cells. Blood: H. B., 80 to 68; R. B. C., 4,960,000 to 4,100,000; W. B. C., 6,750; polys., 53 to 82.5; lymphs., 47 to 17.5; Wassermann negative. The sputum was positive for tubercle bacilli three times. Spinal fluid: 25 cells, 90 to 98 per cent small lymphocytes. Acid-fast organisms found in smear. Colloidal gold, .0012221000. Chemistry: Sugar, 40 to 35. Total protein, 85 and 60.

Roentgen-ray Diagnosis.—Advanced pulmonary tuberculosis.

Anatomic Diagnosis.—Tuberculous cerebrospinal meningitis. Pulmonary tuberculosis, bilateral. Miliary tuberculosis of small intestines, spleen and mesenteric nodes with tuberculous ulcerations of the large intestine. Fibrous adhesions of pleura, bilateral. Fibrous adhesions of pericardium.

Gross Pathology.—External appearance normal. The peritoneal cavity showed adhesions of the omentum about the gall bladder, spleen and to the diaphragm. On the right side of the chest there were adhesions about the apex. The hilus on the left was obliterated by thick fibrous cicatrices. The right lung weighed 525 gms. The upper lobe showed caseous nodules 1 cm. in diameter, dark red and firm. The left lung weighed 712. It was firm throughout. There were many caseous spots in the entire upper and lower lobes. There was a cavity in the upper, 2 cm. in size, filled with bloody and necrotic material. The lower ileum showed several shallow ulcerations. The largest, 6 by 2 cm. The large intestine showed small ulcerations in the cecum, ascending, transverse and entire descending colon. There was slight edema of the pia and arachnoid. The lateral ventricles were distended with slightly cloudy fluid.

Bacteriology.—Antemortem sputum was positive for tubercle bacilli. The spinal fluid was negative. The fluid from the cysterna magna was positive for tubercle bacilli. The postmortem heart blood contained B. Coli, B. Botulinus (1 cc. 1/10,000 filtrate killed a guinea pig in less than 24 hours) and B. Welchii.

Microscopic Examination.—The lungs showed thickened pleura with solid caseous areas and cavitation, i. e., typical tuberculosis. Special stains revealed acid-fast bacilli. The mediastinal nodes were caseated and showed giant cells. The larynx, tongue and epiglottis were normal. The lateral and posterior portions of the arytenoids showed miliary tubercles. The mucosa was almost

Fig. 1. Autopsy 754. There is a tubercle beneath the inner surface of the larynx and it shows little surface epithelium intact. The giant cell is surrounded by epithelioid cells and extensive lymphocytes. The lymph capillaries are prominently distended.

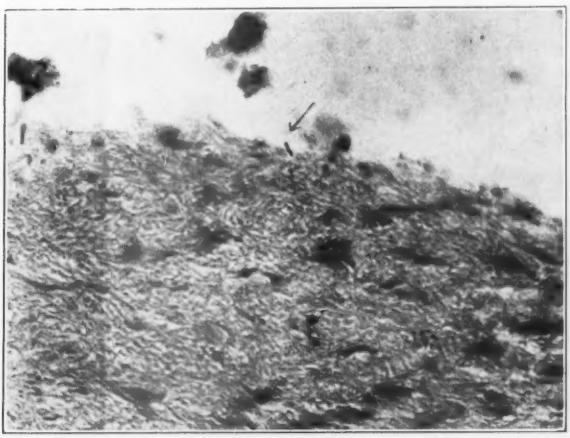

Fig. 2. Autopsy 754. The bacillus tuberculosis is lying deep in the submucosa of the larynx where the tissue has been separated, forming an open crypt, but on one side the bacillus is well situated in the fibrous tissue near the surface of the separation.

completely intact and very vascular. The small intestines showed slight ulceration and mucocaseation with giant cells in the submucosa. The large intestines showed destruction of the mucosa widening into the submucosa with caseation and giant cells. The spleen and mesenteric nodes showed miliary tubercles. The meninges were edematous and congested with plasma cells. There were small tubercles with slight caseation. There was necrosis about the vessels with red blood cells, lymphocytes and plasma cells. The cerebellum was infiltrated on the surfaces and in the crypts with small atypical tubercles. The spinal cord was infiltrated, edematous and congested. Acid-fast bacilli were found in the meninges by special stain.

The epiglottis was normal. The tongue sections were normal. The right arytenoid, near the esophageal wall, was lined by stratified squamous epithelium and was completely intact. The inner laryngeal surface had the columnar cells, mostly intact, even where there were occasional small miliary tubercles with slight caseation and giant cells were found. The submucosa had some edema and an increase in vascularity. The posterior arytenoid margin showed one tubercle and slight ulcerations which were more superficial than on the right side. The left arytenoid margin showed no tubercles, but had edema and slight infiltration of plasma cells and lymphocytes. By special stain a few single acid-fast bacilli were found in the mucosa of the larynx.

Comment.—The patient did not have clinical evidence of laryngeal tuberculosis before death, but did have miliary tubercles in the larynx postmortem.

Autopsy 758.—Mr. P., age 37, male, admitted to the National Jewish Hospital for Consumptives, first, March 8, 1927, discharged October 3, 1928; readmitted June 26, 1929; died November 25, 1929. Occupation, salesman. The duration of pulmonary tuberculosis was twelve years. The patient had been moderately hoarse for four years. There were no symptoms referable to the larynx during the first residence. The laryngoscopic examination showed a moderate degree of chronic infiltration of both arytenoids and vocal bands, with partial immobility of the left vocal cord.

Physical Examination.—This disclosed evidence of far advanced, active, bilateral pulmonary tuberculosis involving the upper halves of both lungs, with cavitations in both upper lobes. During the second residence the symptoms were persistent dryness of the throat and slight pain on swallowing. The laryngoscopic examination did not disclose any noteworthy changes from those elicited during the first residence, except a granular appearance of the interarytenoid space, which was covered with mucopus, not readily removable with applicators. During the second residence there were marked digestive disturbances, and, in addition, constipation alternating with diarrhea.

Clinical Diagnosis.—Far advanced, bilateral, fibro-ulcerative, pulmonary tuberculosis. Chronic myocarditis with partial decompensation. Chronic incipient tuberculous laryngitis and tuberculous enterocolitis. The terminal manifestations were those of progressive right heart dilatation.

Anatomic Diagnosis.—Pulmonary tuberculosis with cavitation, bilateral. Cardiac dilatation and hypertrophy. Generalized anasarca with pulmonary edema. Hydrothorax left. Hydropericardium. Ascites. Edema of lower extremities. Chronic passive congestion of liver. Healed surgical scar in right lower quadrant. Surgical absence of appendix.

Gross Pathology.—Emaciation. Edema of lower extremities. The peritoneal cavity contained about 400 c.c. of clear fluid. There were many prominent lymph nodes in the mesentery. The liver was 7 cm. below the costal margin on the right. The entire right and most of the left pleural cavities were obliterated by adhesions. Pocketed cavities on the left were filled with fluid. The heart was enlarged. The wall of the right ventricle was as thick as that of the left. The chambers were all enlarged. The laryngeal surfaces were all smoothly covered by mucosa with no evidence of congestion or edema. Both vocal cords had fairly large, white nodular thickenings near the posterior insertions. Both upper lobes of the lungs were extensively cavitated. The remaining lobes were aerated and congested.

Microscopic Examination.—The lungs showed cavitation, caseous nodules, fibrosis, congestion and edema. The mucosa of the epiglottis was entirely intact and beneath it, over the posterior

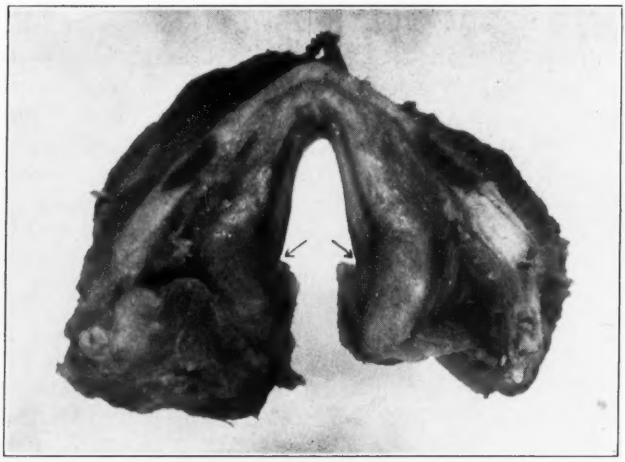

Fig. 3, Autopsy 758. The larynx shows pale nodular enlargements upon both vocal cords near the posterior insertion.

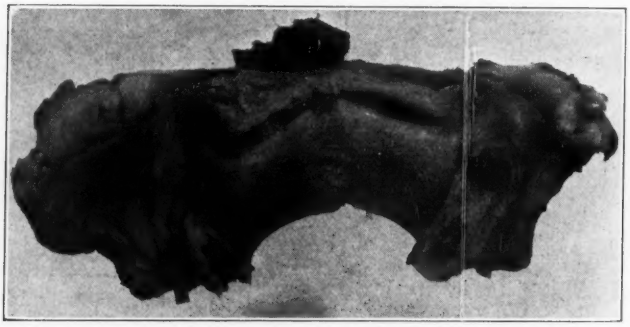

Fig. 4, Autopsy 758. The larynx shows pale nodular enlargements upon both vocal cords near the posterior insertion. Same specimen as Fig. 3.

Fig. 5, Autopsy 758. The tissue from the base of the tongue shows the epithelium intact and a large lymph follicle closely under it having an early tubercle with slight central caseation and many epithelioid cells.

surface, were scattered plasma cells and lymphocytes with little edema and no increase in vascularity. The mucosa of the base of the tongue on both sides was intact, and along the sides there were many plasma cells which extended slightly into the lymphoid tissue, numerous follicles of which were present. In one follicle on the left side of the tongue was a small caseous tubercle with epithelioid cells. Tubercle bacilli were not found in sections of the tongue with special stain. The mucosa of the left vocal cord was almost completely intact. Upon the surface of the pyriform sinuses there was purulent exudate. Even about this, where the mucosa was broken, there was no inflammatory reaction. In the stroma of the right cord there was a large, extensively dense, fibrous nodule. The right and left arytenoid margins showed edema and infiltrated plasma cells. There were lymphocytes on the laryngeal side, but much less on the esophageal side. In no place was the mucosa broken. By special stain the tissues from the base of the tongue, epiglottis and arytenoids did not show the presence of acid-fast bacilli. Above the left vocal cord the exudate and desquamating epithelium contained many acid-fast bacilli. There were none in the intact epithelium.

Comment.—This patient had an incipient tuberculous infiltration of the arytenoids prior to death, but we were not able to find acid-fast bacilli microscopically except above the left vocal cord. He probably had a pachydermia from chronic cough and not a true tuberculous infiltration. Many patients with pulmonary tuberculosis have a chronic laryngitis produced by prolonged irritation from a chronic cough but not a tuberculous laryngitis. This is often a pachydermia of the arytenoids and interarytenoid sulcus.

Autopsy 711—Mr. M., National Jewish Hospital for Consumptives, age 25, single, male. Occupation, bookkeeper. Admitted May 13, 1928; died August 8, 1929. Cause of death given as pulmonary tuberculosis and suspected enterocolitis, because of clinical manifestations pointing to such. The duration of the pulmonary tuberculosis was six and one-half years.

In the spring of 1925, three years before admission, the patient began complaining of slight pain upon swallowing, the sensation of a lump in the throat and some hoarseness. The symptoms, according to the patient, subsided after the larynx had been cauterized during the subsequent eight months. He had no laryngeal symptoms until shortly before his admission. These symptoms were dryness, occasional pain, not radiating to either ear, and slight hoarseness. After the epigiottis was cauterized on two different occasions, practically all the symptoms subsided, and during the entire residence the patient did not complain of any symptoms referable to the larynx.

Physical Examination.—This showed far advanced, active, pulmonary tuberculosis involving the entire left lung and part of the right upper lobe, complicated by chronic laryngeal tuberculosis. During the entire residence the patient pursued an active and slowly progressive course.

Laboratory Examination.—The urine was negative. The blood always showed a high leucocyte count, ranging from 24,200 to 38,800, with 80 to 90 per cent polymorphonuclear leucocytes. The sputum was positive for tubercle bacilli at all examinations.

After admission, May 24, 1928, examination of the larynx showed considerable infiltration of the interarytenoid space. The epiglottis was notched at the center with the edges ulcerated.

Laryngeal Examination, October 18, 1928.—All the epiglottidean ulcerations had healed under treatment, presenting white scarred areas where the cautery had been applied. There was considerable infiltration of the interarytenoid space.

Anatomic Diagnosis.—Pulmonary tuberculosis with cavitation, bilateral. Tuberculous ulceration of the large intestine. Tuberculous appendicitis. Healed tuberculosis of epiglottis. Partial removal of epiglottis by cauterization. Tuberculous laryngitis. Central necrosis of liver.

Gross Pathology.—Fibrous pleural adhesions, bilateral. Cutaneous scar of right thigh. Firm fibrous adhesions about the transverse colon, gall bladder and pyloris of the stomach. The mediastinum had the right pleural wall 3 cm. to the left of the median line. The left pleural cavity was completely obliterated, and the right showed many scattered adhesions. The right lung was greatly distended and large, having irregularly distributed small firm spots throughout, which upon cut surfaces was caseous. There were cavities in the apex. The left lung was small, firm and not aerated. The cut surfaces were dark red and firm with many distended, fibrosed bronchioles. Many cavities occupied the apex. The epiglottis had the upper edge missing, so that the edge was irregularly diagonal and thick, but the entire surface over this appeared to be smoothly covered with mucosa. The arytenoids and rima of the larynx appeared thick, soft and covered by smooth mucosa. The appendix was matted to the posterior pelvic brim and appeared irregularly swollen and dark red. The large intestines had numerous long and narrow ulcerations in the cecum, ascending and transverse portions.

Microscopic Examination.—Typical tuberculous lesions. The large intestines had chronic inflammatory ulcers with multinuclear giant cells and caseation in the submucosa. The appendix showed giant cell tubercles and caseation in the submucosa with little loss of the overlying glandular mucosa. The liver showed central necrosis of the lobules.

The base of the tongue showed intact mucosa and beneath it a very marked edema with numerous large and prominent lymph capillaries. In the superficial stroma there were some plasma cells and lymphocytes. In most all of the lymph follicles there

Fig. 6. Autopsy 711. The gross picture of the throat organs showing a healed epiglottis, following the use of the cautery, and thickening of the arytenoids.

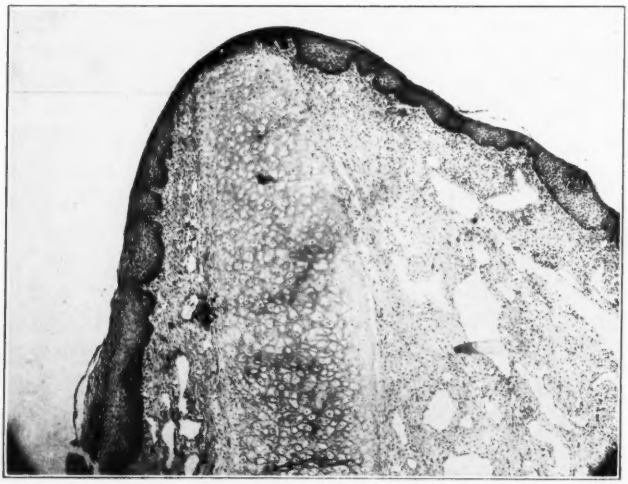

Fig. 7. Autopsy 711. A microscopic illustration of the epiglottis showing the site of healing over the tip by a thin overgrowth of stratified epithelium separated but little from the tip of the cartilage. Elsewhere the stroma is thickened by interstitial and lymph capillary edema.

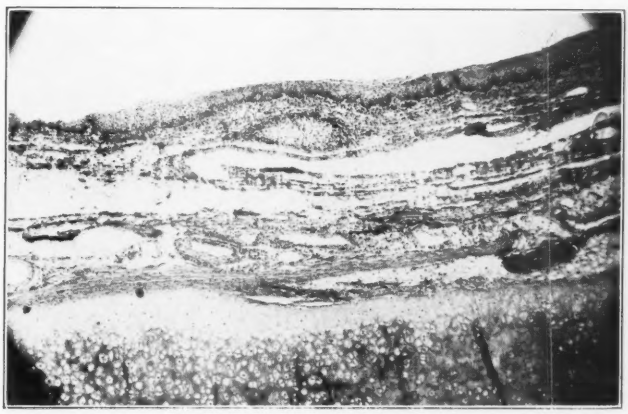

Fig. 8, Autopsy 711. The lower power magnification of the epiglottis in the lower anterior wall. The surface epithelium is intact. Near it, in the stroma, there is a giant cell surrounded by epithelioid cells and a nest of lymphocytes which latter are scattered slightly in the surrounding stroma. The lymph capillaries are prominently distended forming a wide stretch between the epithelium and the cartilage.

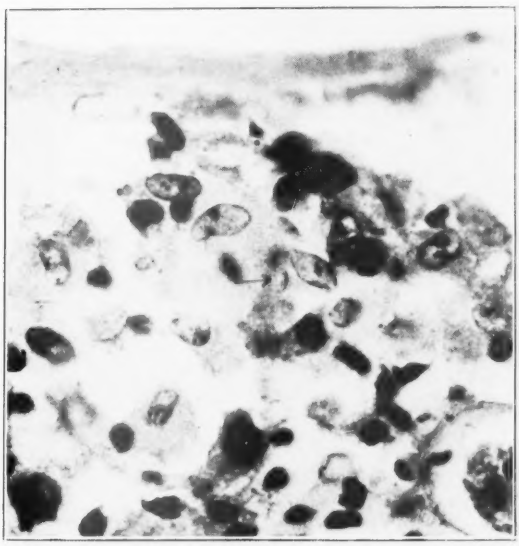

Fig. 9, Autopsy 711. The oil immersion shows acid-fast bacilli in the tubercles in the lower anterior wall of the epiglottis.

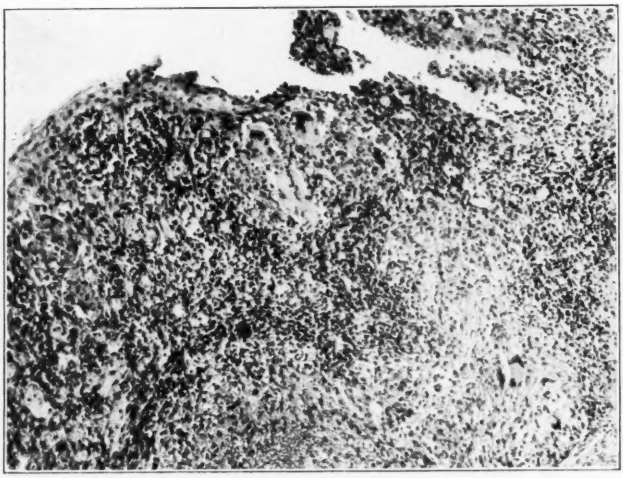

Fig. 10. Autopsy 711. There is ulceration in the surface epithelium and underlying tubercles, extending deep in the submucosa of the posterior wall of the larynx just below the rim.

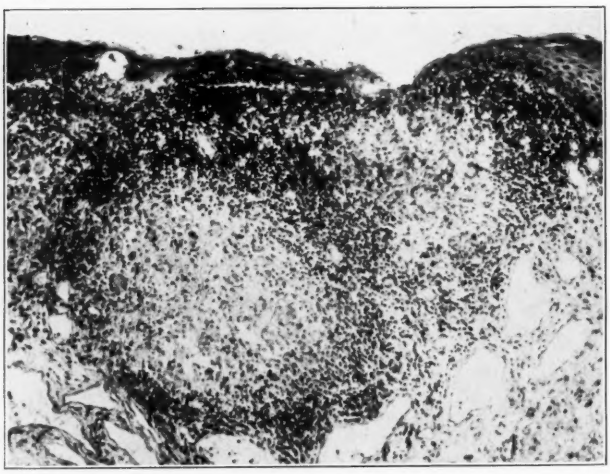

Fig. 11. Autopsy 711. There is thinning and approaching ulceration of the mucosa in the anterior wall of the esophagus at the upper rim of the larynx. This shows tubercles, including multinuclear giant cells, epithelioid cells surrounded by lymphocytes and below this are prominent, distended lymph capillaries.

were small areas that were apparently early tubercles, comprised of caseation with epithelioid cells but no giant cells. The surface mucosa of the epiglottis had a slight break in continuity about the middle of the posterior surface. About this small ulceration were clusters of lymphocytes with epithelioid cells and a few multinuclear giant cells extending for some depth into the stroma. Figure 9 shows an acid-fast bacillus in the lower anterior wall of the epiglottis. At the upper end the stratified epithelium was very thin and separated from the cartilage by only a very thin strand of fibrous tissue stroma at the site of the healed cauterization at the tip of the epiglottis. Except at this point the submucous stroma was thickened by edema and prominently distended lymph capillaries. In the lower anterior wall there was in the stroma a single giant cell tubercle. One of the distended lymph capillaries was almost filled with a cluster of polymorphonuclear leucocytes.

Comment.—This patient had had laryngeal tuberculosis confined chiefly to the tip of the epiglottis. While this healed, following cauterization, he had acid-fast bacilli in the lower anterior wall of the epiglottis.

Autopsy 691.—Mr. S., age 32, male, single; occupation, salesman. Admitted to the National Jewish Hospital for Consumptives, November 29, 1928; died June 29, 1929. The duration of the pulmonary disease was three and a half years. There were no symptoms referable to the larynx until three or four weeks prior to death. The principal symptoms then complained of were marked dryness and slight pain on swallowing.

Physical Examination.—This showed evidence of a far advanced, active, bilateral pulmonary tuberculosis, with pronounced cavitation in the left apex. There were marked intestinal symptoms pointing to the possibility of tuberculous enterocolitis.

Laboratory Examination.—The sputum was positive at all examinations. All other laboratory findings were essentially negative.

Laryngoscopic Examination.—This disclosed a moderately thickened epiglottis, but because it was of the low, overhanging type a satisfactory view of the laryngeal aspect could not be

obtained. For the same reason the interior of the larynx could not be satisfactorily examined. There was no aggravation of the laryngeal symptoms, notwithstanding the rapid progression of the pulmonary disease.

Gross Pathology.—There was a small decubitis ulcer over the sacrum. There was a large defect in the anus, together with a firm polypoid tumor. There were numerous small mesenteric lymph nodes. The liver was much enlarged. The left pleural cavity was entirely obliterated and the right had dense adhesions at the apex. The pericardial cavity contained about 100 cc. of clear fluid. The right pleura was thickened. There was a large cavity at the apex of the right lung. The middle lobe contained numerous caseating areas. The left pleura was thick. There were cavities throughout the upper lobe. The lower border of the lower lobe was emphysematous.

The posterior surface of the epiglottis had several ulcerated areas, and there was a small ulcer on the left false cord. The mucosa over the posterior surface of the epiglottis was pale, granular and thickened. In the cecum and first eight inches of the ascending colon there were numerous shallow ulcerations.

Microscopic Examination.—The lungs showed typical tuberculous cavitation and caseation with giant cells about caseous areas and surrounded by fibrous tissue. There was atelectasis and emphysema in different places. The large intestine showed a small ulcer. There was a hemorrhoidal polyp at the anus. There was a large amount of amyloid in the spleen and liver.

The epiglottis had two extensive areas, with loss of mucosa, on the posterior surface and also in part of the stroma. Upon the surface of this ulceration was a collection of polymorphonuclear leucocytes. In the deeper tissues there were lymphocytes, epithelioid cells and a few polymorphonuclear cells near the surface. Still deeper was a small miliary tubercle. In one place the ulceration was so deep that it involved the cartilage. The stroma in general had considerable increase in fibrous tissue with tubercles, giant cells, many scattered epithelioid cells and lymphocytes. Special stains showed many acid-fast bacilli in the stroma and in the ulcers. The laryngeal mucosa appeared normal. The support-

ing tissue was fairly dense and slightly vascular. In one pocket, on the surface, was a small collection of purulent exudate. No tubercle bacilli were found. A small portion of the surface mucosa of the vocal cords was absent at a point slightly below the true vocal cords, and here there was a little exudate on the surface. The stroma had increased thickness, due to the presence of epithelioid cells, lymphocytes and a few plasma cells. The false cord on one side had a tubercle just under the surface epithelium. The true vocal cord on the same side had the stroma very greatly thickened by dense fibrous tissue, most of which was hyalinized. There were many tubercle bacilli in the stroma about the ulcer below the vocal cord.

Diagnosis.—Chronic pulmonary tuberculosis with cavitation and tuberculous bronchopneumonia. Tuberculous laryngitis with ulceration. Plural adhesions, bilateral. Tuberculous ulceration of cecum and colon. Fistula of anus.

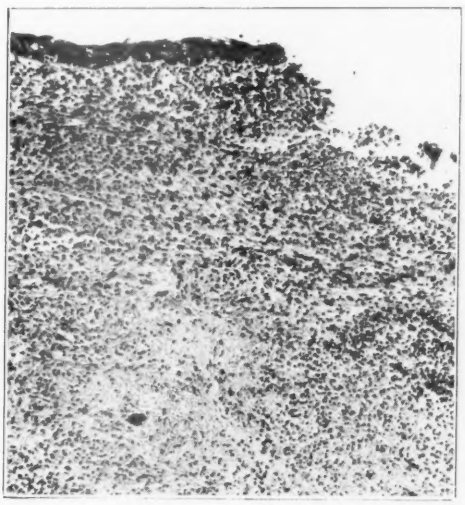

Fig. 12. Autopsy 691. The margin of an ulcer in the middle of the posterior wall of the epiglottis, in the surface of which is slight purulent exudate. Beneath this there are many epithelioid cells and lymphocytes. In the deeper part of the stroma are seen giant cells.

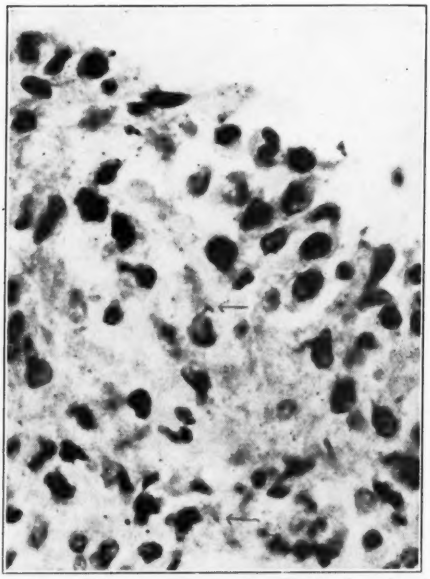

Fig. 13. Autopsy 691. An oil immersion illustration showing acid-fast bacilli in the submucous stroma of the epiglottis.

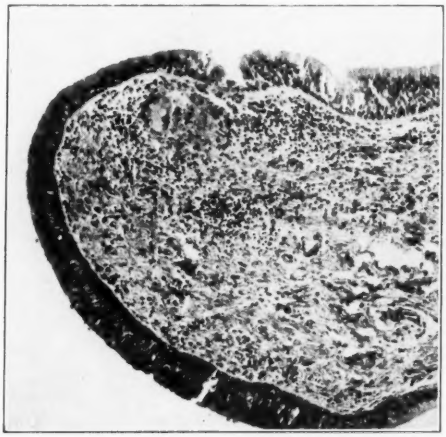

Fig. 14. Autopsy 691. The false vocal cord has intact epithelium and a large multinuclear giant cell in the submucous stroma, which is loosely edematous and has some epithelioid cells with many scattered lymphocytes.

Autopsy 825.—Mr. M., age 29, American, male, admitted to the National Jewish Hospital for Consumptives, first, March 17, 1927, and discharged June 2, 1929; readmitted March 22, 1930: died April 1, 1930. The duration of his illness was three years and nine months. On admission the pulmonary tuberculosis was equal on both sides. Later it became more pronounced on the left side. Pneumothorax was later instituted on that side. Following the pneumothorax the patient did not do well and later developed an effusion. The pneumothorax was discontinued. After a number of months the patient, although not doing well, decided to go home.

During the first residence the patient had no symptoms referable to the larynx except for an occasional complaint of dryness and some irritation described as a peculiar tickling sensation which gave rise to cough. The laryngoscopic examination at the time showed no definite pathology except for a moderate secondary anemia of all structures.

About two months prior to the second admission the patient began to complain of gradually increasing pain, radiating to both ears, increasing salivation, moderate dysphagia and dysphonia. The laryngoscopic examination at the second admission showed the entire epiglottis to be considerably infiltrated, but with no ulceration. Both arytenoids were likewise infiltrated. The ulcerations were noted in the interarytenoid space and on the left vocal cord.

The patient was readmitted to the National Jewish Hospital for Consumptives March 22, 1930. He gave a history of having developed diarrhea in September, 1929, and a tuberculous laryngitis in December, 1929. In January, 1930, the patient also developed an otitis media. Examination on the second admission showed bilateral tuberculosis with cavitation on both sides. Due to extreme emaciation it was difficult to elicit any other findings. A few days after admission the patient developed edema of the lower extremities. The diarrhea became worse; likewise the laryngitis. The patient died within a week after admission.

Laboratory Examination.—Albumin was present in the urine, a few hyaline casts and many white blood cells. The sputum was always positive for tubercle bacilli. The hemoglobin on the

second admission was 72 per cent. The red blood count was 3,820,000; white count, 11,200, and polys, 86. The Wassermann was negative. At the second admission, the roentgen-ray showed extensive fibrosis and inflammatory infiltration with numerous cavities. On the right side there was a very large cavity from the apex to the third rib and infiltration down to the base.

Diagnosis: Chronic pulmonary tuberculosis with bilateral cavitation and tuberculous bronchopneumonia. Tuberculous enterocolitis. Tuberculous laryngitis. Fibrous adhesions of pleura with obliteration, bilateral. Fibrous adhesions of pericardium with obliteration.

Gross Pathology.—The liver was 6 cm. below the xyphoid cartilage and 4 cm. below the costal margin. Over the small intestines there were many radiating areas of congested blood vessels. The peritoneum over these was finely granular and dull. There were many prominent mesenteric lymph nodes. The pleural cavities were obliterated, except at the basal surface on the right. The pericardial cavity was completely obliterated by firm fibrous adhesions.

The epiglottis was soft and thickened. The arytenoids were dark red and thickened. The vocal cords were thick and soft. There were very small ulcers over the posterior laryngeal wall and just above the posterior end of the left false cord.

The upper lobes of both lungs were extensively cavitated. The rest of the lungs were filled with small caseous nodules. In the mediastinum several nodes showed caseation on section. The upper jejunum had a few ulcerations. The rest of the jejunum and the whole of the ileum showed most all of the mucosa lost by large deep ulcers. There was no complete perforation. The appendix was thickened and dark red. The large intestines had very many large, irregular ulcers throughout.

Microscopic Examination.—The heart showed thick pericardium formed by loose, edematous and very vascular fibrous tissue with little infiltration. The lungs showed caseation, tubercles and emphysema. The mediastinal nodes showed tubercles and caseation. The small intestines showed large ulcerations. The large intestines showed a shallow ulceration with isolated caseation and numerous tubercles in the submucosa. The mesenteric nodes

showed tubercles and amyloid degeneration. The spleen and liver showed much amyloid.

The base of the tongue had intact mucosa. The stroma showed slight edema and scant infiltration of any cells. In a few lymph follicles were indications of early tubercles. The epiglottis had the mucosa almost entirely intact. A small area was missing at the middle of the posterior surface. The stroma was markedly increased in thickness, mostly by the presence of many epithelioid cells, lymphocytes and a few plasma cells. These were imperfectly formed tubercles, some of which showed slight caseation and an occasional giant cell. The stroma had an increase in loose fibrous tissue, and the lymph capillaries were numerous and large. Over the esophageal and laryngeal surfaces of the arytenoid margin there was an extensive loss of the surface epithelium. The surfaces were rough and formed by loose fibrous tissue with many epithelioid cells, lymphocytes and polymorphonuclear cells. The latter were on the most superficial portion. The mucous stroma was increased in thickness by fibrous and vascular tissue proliferation and edema. This affected the larynx, including the false vocal cords, to the level of the true cords at their posterior insertion and the vocal cords, with excoriation of the surface epithelium at the most posterior portion. The epithelium was intact over the remainder of the vocal cords. The entire cords had the stroma made very thick by edema and increased fibrous tissue, through which were many epithelioid cells, lymphocytes and, at the ulcerated portion, polymorphonuclear cells. Under the epithelial surface were occasional tubercles. The false vocal cords had tubercles.

Special Stain.—Acid-fast bacilli were present in myriads, scattered through the stroma of the epiglottis the full length of the anterior and posterior surfaces; through the stroma of the ulcerated wall of the larynx and through the stroma of the vocal cords. There was a very large number at the middle portion of the cord where the epithelium was intact. None were observed in the tongue.

Comment.—This patient had definite clinical laryngeal tuberculosis before death, for several months, and definite postmortem evidence of the same, both gross and microscopic.

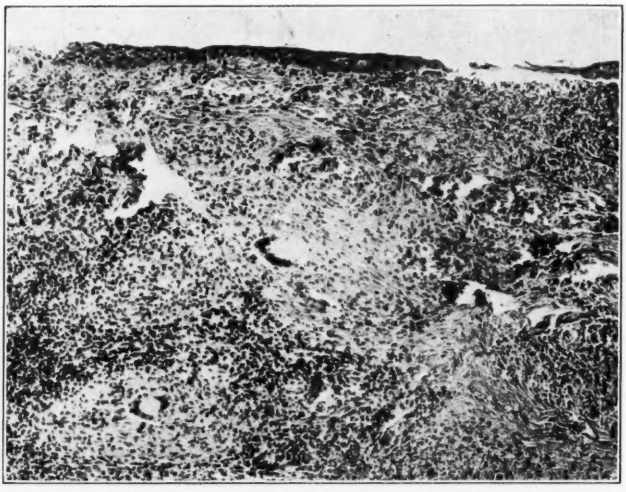

Fig. 15, Autopsy 825. A section taken toward the side of the epiglottis shows thinning and slight loss of the surface stratified epithelium, beneath which are tubercles with some fibrosis. This is in the lower portion of the anterior wall of the epiglottis.

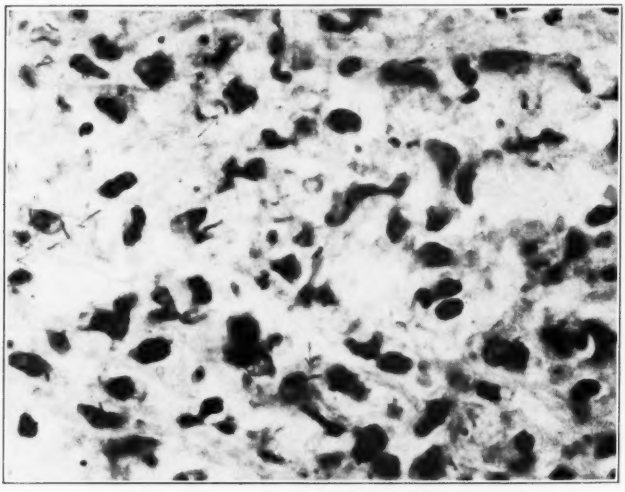

Fig. 16, Autopsy 825. An oil immersion illustration shows a large number of acid-fast bacilli, which were distributed throughout the stroma on both the anterior and posterior surfaces of the epiglottis.

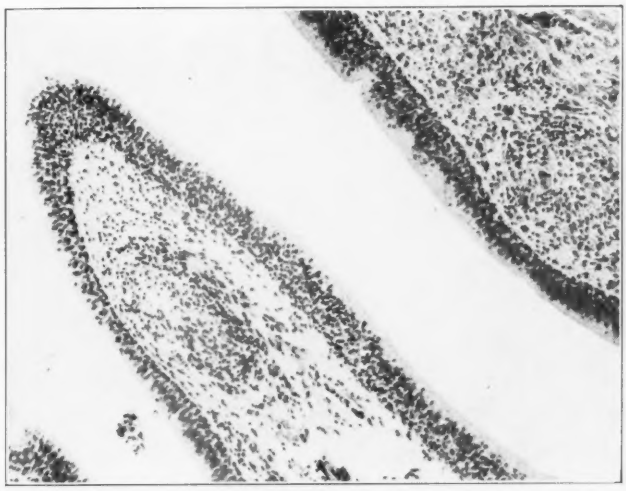

Fig. 17. Autopsy 825. An early tubercle formation in a fold of the false vocal cord.

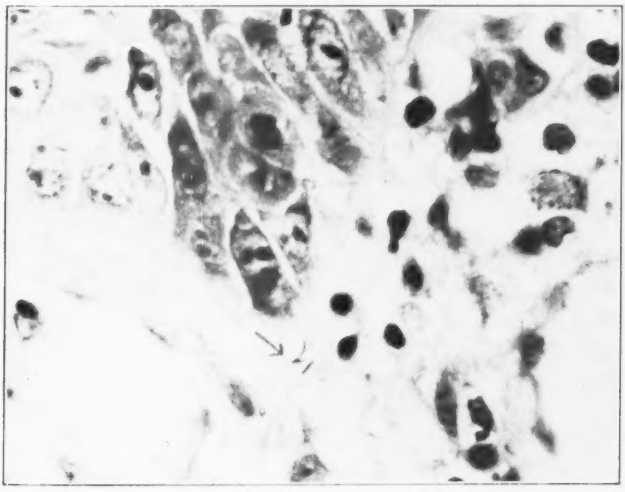

Fig. 18. Autopsy 825. An oil immersion picture taken at the tip of the true vocal cord showing acid-fast bacilli in the basement membrane, below the surface epithelium, with the latter intact.

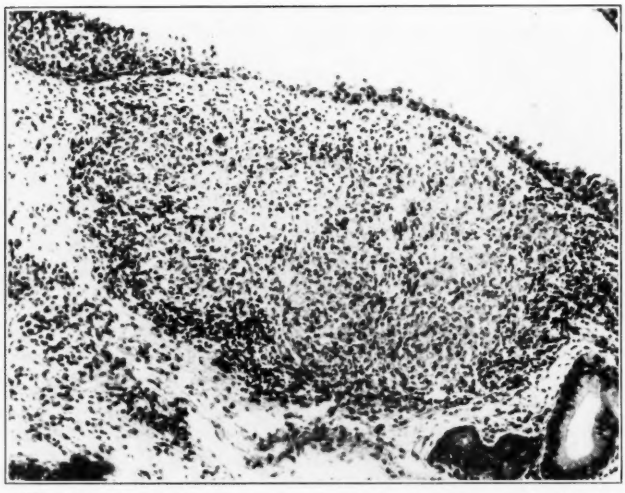

Fig. 19. Autopsy 825. There are tubercles in the upper surface of the true vocal cord, lymphocytes, epithelioid and plasma cells, distant from the tip.

SUMMARY AND CONCLUSIONS.

Autopsy 827 revealed only a low grade chronic inflammation of the larynx, but no evidence of tuberculosis before or after death. Photographs have been omitted because they have no particular interest in this study.

Photographs 1 and 2 show tuberculous lesions. Number 1 shows a giant cell and Number 2 one tubercle bacillus. This was deep in the tissue of the submucosa. In making the section the tissue formed a crack along the line of which separation the acid-fast bacillus lay. These are from autopsy 754.

Photographs 3, 4 and 5 also show tuberculous lesions. Number 5 shows a large tubercle with beginning caseation. These are from autopsy 758.

Photographs Numbers 6, 7, 8, 9, 10 and 11 show many lesions of tuberculosis, such as a giant cell surrounded by epithelioid cells and lymphocytes in 8, acid-fast bacilli in 9, ulceration in 10, with underlying tubercles, and in 11 tubercles, including multinuclear giant cells and epithelioid cells. These are from autopsy 711.

Photographs 12, 13 and 14 show gross and microscopic lesions of tuberculosis and acid-fast bacilli in 13. These are from autopsy 691.

Photographs 15, 16, 17, 18 and 19 all show chronic inflammation, tubercles in 15, 17 and 19 and acid-fast bacilli in 16 and 18. These are from autopsy 825.

COMPARATIVE TABLE OF LARYNGEAL LESIONS.

Autopsy No.	Epiglottis	Base of Tongue	Arytenoids	Vocal Cords
827	Edema and chronic inflammation	Neg.	Neg.	Neg.
754	Neg.	Neg.	Giant cell and epithelioid cells	Tubercle bacillus
758	Neg.	Tubercle and epitheliloid cells	Neg.	Tubercles
711	Giant cell and epitheliloid cells. Tubercle bacillus.	Neg.	Ulceration and giant cell tubercles	Neg.
691	Ulceration Tubercle bacilli Tubercle	Neg.	Infiltration	Giant cell and epithelioid cells
825	Tubercle bacilli Giant cell	Neg.	Neg.	Early tubercle

Two of the patients had symptoms of laryngeal tuberculosis before death and four did not. However, only one, autopsy number 827, failed to show microscopic lesions, thus proving our contention that patients suffering from pulmonary tuberculosis must have microscopic lesions of tuberculosis for some time before subjective and objective symptoms become manifest. The time to prevent laryngeal tuberculosis is by an early diagnosis of the pulmonary disease and early treatment. "An early discovery means early recovery," usually.

There were no clinical symptoms of laryngeal tuberculosis in autopsy cases 754, 758 and 691, but lesions were found in the larynx at postmortem.

There were subjective and objective symptoms in the larynx in autopsy case 711 and tuberculous lesions postmortem.

Clinical symptoms, subjective and objective, with gross and microscopic lesions, were all present in autopsy case 825.

The photographs used to illustrate this paper have not been selected at random, but they have been selected because they illustrate the typical microscopic tubercles and acid-fast bacilli found in five of the six patients studied before and after death.

We have not found, after a careful search of the literature of otolaryngology and pathology, that similar work has been reported.

Fetterolf[1] reported the results of his gross postmortem studies, based on 100 cases, before this society (American Laryngological Association), in 1914, in a very comprehensive paper. He says "the present report will deal only with the gross appearances, the microscopic findings being left for a future communication." The chief of otolaryngology, Dr. Robert Levy, tells us he wrote Dr. Fetterolf about the microscopic study of his specimens, but learned from him that it was not completed. Dr. Levy, in 1925, started to study the histopathology of laryngeal tuberculosis, but did not get to finish because of ill health in his family. We did not know about this until a few weeks ago. Levy[2] suggested such a study in 1906.

There are, of course, many reports of the histopathology of laryngeal tuberculosis, when gross lesions were known to exist prior to death. Our investigation was undertaken to show that microscopic tubercles do exist, antemortem, in patients in whom the chief cause of death was pulmonary tuberculosis, but who did not have demonstrable laryngeal lesions during life.

The criticism may be offered that we have too few cases. We have only six. The University of Colorado General Hospital does not take very many tuberculous patients, because if it did we would soon have nothing else for teaching purposes. Five of the cases are from the National Jewish Hospital for Consumptives and one from Colorado General Hospital, but the postmortem specimens were all studied at the University of Colorado School of Medicine.

PHYSICIANS' BLDG.

BIBLIOGRAPHY.

1. Fetterolf, George: The Larynx in One Hundred Cases Dying of Pulmonary Tuberculosis. Trans. Amer. Lar. Assn., 1914, p. 258.

2. Levy, Robert: Surgical Treatment of Tuberculosis of the Upper Air Passages and the Ear. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1906, p. 587.

LXXIX.

LARYNGECTOMY, SINGLE STAGE—OPERATIVE RESULTS.*

HENRY BOYLAN ORTON, M. D.,

NEWARK, N. J.

"The control of infective material normally present in a region has taken a large part in certain great surgical advances." (Trotter.[1])

Our late Dr. Mackenty[2] did more to reduce infections in laryngectomy than any other, and he has succeeded in associating his name inseparably with some names which will last as long as our language. (Macaulay.) I cannot help digressing for a moment to quote what Dr. Harmon Smith so beautifully said of him, "His sudden departure was like a flower touched by an untimely frost, while its bloom and fragrance still responded to the morning sun."

The first step in laryngectomy must be proper exposure; secondly, the control of infection, either in the lung or extrapharyngeal tissues, so fatal in surgery of this area. Careful operative technic will reduce that risk but cannot exclude it with certainty.

My procedure in general follows the technic of Gluck[3] and Mackenty, with emphasis on the dissection by planes; by so doing we obtain that proper exposure, we can isolate and control the bleeding, and reduce to a great extent the contamination of the wound by pharyngeal contents.

This winter, with the kind assistance of Dr. Mosher in his laboratory, I prepared dissections from which plaster casts were made showing the procedure.

A "T" incision is made, the vertical portion extending from the hyoid bone to the suprasternal notch; the horizontal portion extending along the upper border of the hyoid bone from one anterior border of the sterno-cleido-mastoid muscle to the other.

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*Presented before the American Laryngological, Rhinological and Otological Society, May, 1932, Atlantic City.

Fig. 1. Laryngectomy—"T" Skin Incision.

1. Vertical—From hyoid bone to suprasternal notch.

2. Horizontal—Along upper margin of hyoid bone to anterior edge of sternomastoid.

3. Superficial fasciæ.

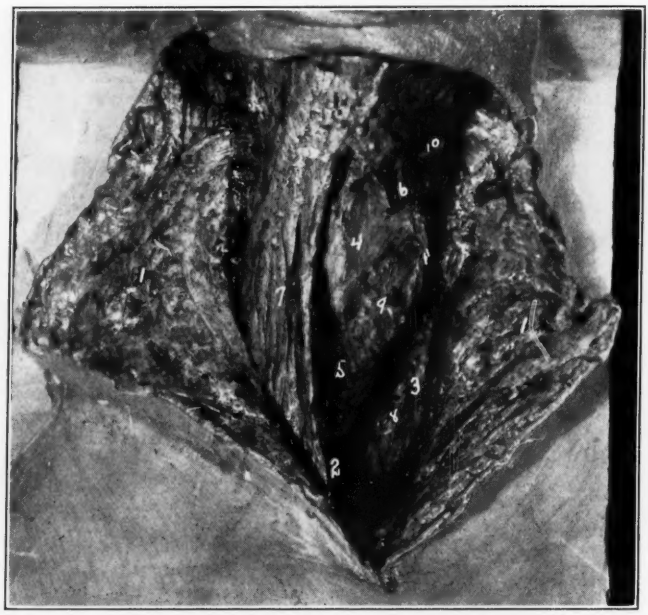

Fig. 2. Laryngectomy—Skeletonization of Larynx.

1. Skin flaps.
2. Trachea.
3. Thyroid gland.
4. Thyroid cartilage.
5. Cricoid cartilage.
6. Thyrohyoid muscle.
7. Sternohyoid muscle.
8. Cricothyroid artery.
9. Sternothyroid muscle.
10. Superior laryngeal nerve, artery, vein.
11. Inferior constrictor muscle.

These skin flaps are dissected outward by means of scissors, thereby making a triangle; the apex at the suprasternal notch, and the base at the upper border of the hyoid bone.

We come back to the superficial fascia covering the sternohyoid muscle. These two muscles are separated in the median line and by blunt dissection with the finger the entire sternohyoid muscle is undermined and separated from the sternothyroid muscle. The sternohyoid muscle is then cut about 1 cm. from its insertion at the hyoid bone and reflected outward. The sternothyroid muscle is cut from its insertion on the thyroid cartilage

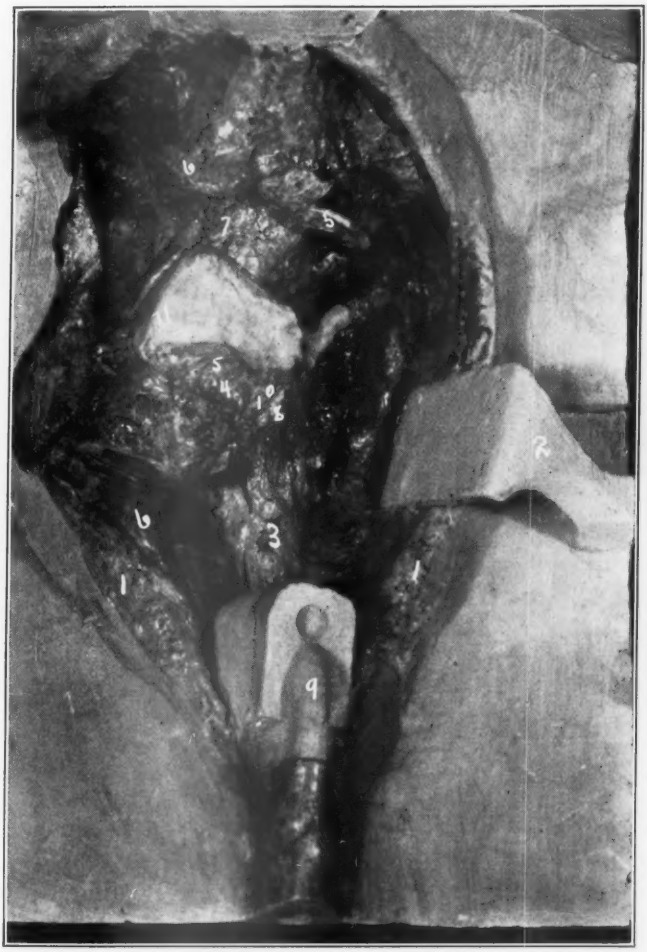

Fig. 3. Laryngectomy—Larynx Amputated From Trachea and Freed From the Esophagus Up to the Arytenoids.

1. Skin flaps.
2. Retractor.
3. Esophagus.
4. Sternothyroid.
5. Thyrohyoid muscle.
6. Sternohyoid muscle.
7. Thyrohyoid membrane.
8. Stylopharyngeus muscle.
9. Anesthesia tube in trachea.
10. Inferior constrictor muscle.
11. Larynx freed from esophagus.

and reflected outward. The thyrohyoid muscle is cut from its attachment on the thyroid cartilage and reflected upward. The thyroid gland, if it is in your way, is clamped in the median line, cut and ligatured. The cricothyroid artery is identified, clamped, severed and ligatured.

The larynx is now skeletonized, and the inferior constrictor muscle is cut from its attachment on the thyroid cartilage along with the stylopharyngeus and palatopharyngeus muscles, and inserted along the posterior border of the thyroid cartilage. The superior laryngeal vessels and nerve are identified, clamped, severed and ligatured. The trachea is partially severed below the cricoid cartilage, and a needle threaded with silkworm gut is passed through the trachea, acting as a stay, thereby preventing its retraction into the neck. Packing is placed in the lower end of the severed larynx. A flap of mucous membrane is made from the posterior wall of the cricoid cartilage, after the procedure of Lewis; then the trachea is completely severed to the esophagus. The anesthesia at this time is transferred to the tracheal opening, using Tucker's anesthesia tube, around which gauze is tightly packed to prevent any leakage of secretion in the trachea. The larynx is separated from the esophagus from below upward to a point opposite the arytenoids.

The pharynx is opened through the thyrohyoid membrane opposite to the side of the growth and packed with one yard of two-inch gauze. The larynx is then removed with the epiglottis. The wound is inspected and all bleeding points are ligated. The pharyngeal opening is then brought together by means of interrupted silk or thread sutures. Before the last interrupted suture has been tied, the anesthetist removes the packing from the pharynx by way of the mouth, and a nasal feeding tube is inserted through one nostril and guided down the esophagus. The esophagus is then reinforced by two rows of catgut sutures. The severed sternohyoid muscles are brought together over the esophagus and sutured and the entire wound is packed with gauze. The tracheal stay suture is brought out, about one inch below the tracheal opening on the skin, and tied over a lead button to relieve tension.

Fig. 4. Laryngectomy—Larynx Removed, Pharyngeal Opening Closed.

1. Skin flaps.
2. Trachea.
3. Esophagus.
4. Hyoid bone.
5. Sternohyoid muscle.
6. Trachea sutured to skin.
7. Inferior constrictor muscle

Our attention then is directed to the severed trachea, the first cartilaginous ring of which is submucously resected, and then with silk the mucous membrane of the trachea is sutured to the skin.

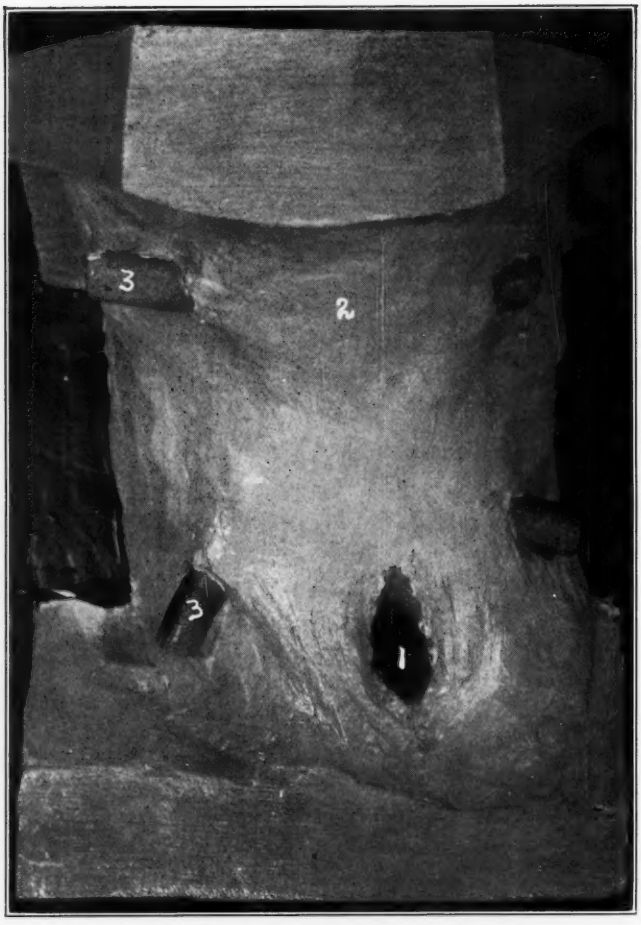

Fig. 5. Laryngectomy—Skin Closure and Drainage.
1. Trachea. *2.* Closure of "T" incision. *3.* Drainage tubes.

Drains are placed at each angle of the transverse "T" incision, and two counter drains are placed laterally on a line just above the tracheal opening. The vertical as well as the horizontal skin incisions are sewed up tight. The laryngectomy tube is wound with gauze impregnated with bismuth paste, fitting the trachea as a cork in a bottle, and secured by tapes around the neck and dry dressing applied. The patient is put to bed in a sitting position.

TABLE No. 1.
CASES OPERATED ON, 32.

	No. of Cases	Private Patients	Hospital Patients	Age
Male	31	19	13	37-76
Female	1	1	0	55

TABLE No. 2.
SHOWING AGE AND SEX AT TIME OF OPERATION.

Age:	31-40	41-50	51-60	61-70	71 and over
Male	2	10	7	8	4
Female	0	0	1	0	0

TABLE No. 3.
SHOWING PATIENTS ALIVE AND WELL.

Case No.	Age at Time of Operation	Sex	Present Age	Period Since Operation
4	72 years	Male	80 years	8 years
5	50 "	"	57 "	7 " 3 mos.
11	50 "	"	55 "	5 "
14	48 "	"	52 " 6 mos.	4 " 6 mos.
16	48 "	"	52 "	4 "
17	66 "	"	70 "	4 "
18	49 "	"	53 "	4 "
20	50 "	"	53 "	3 "
21	70 "	"	73 "	3 "
23	54 "	"	56 " 6 mos.	2 " 6 mos.
24	38 "	"	40 " 3 mos.	2 " 3 mos.
25	54 "	"	56 " 3 mos	2 " 3 mos.
27	60 "	"	62 "	2 "
28	49 "	"	50 " 6 mos.	1 " 6 mos.
29	76 "	"	77 "	1 " 4 mos.
30	60 "	"	60 " 7 mos.	7 mos.
31	46 "	"	46 "	5 "
32	63 "	"	63 "	4 "

TABLE No. 4.

DEATH FROM OTHER CAUSES.

Case No.	Age at Operation	Age at Death	Period After Operation	Cause of Death
22	63 years	65 years	2 yrs. 6 mos.	Apoplexy

TABLE No. 5.

DEATHS FROM MALIGNANCY ELSEWHERE THAN LARYNX.

Case No.	Age at Operation	Time of Onset After Operation	Site of Growth at Time of Operation	Age at Death	Location of Recurrence
1	50 yrs.	6 mos.	Subglottic	50½ yrs.	Glands
2	63 "	13 "	Pyriform sinus	64 "	Glands
7	66 "	8 "	Pyriform sinus	66 "	Glands
8	50 "	1 yr.	Epiglottis	51 "	Tongue
9	60 "	1 " 6 mos.	Arytenoid and aryepiglottic fold	62 "	Glands
12	60 "	2 yrs.	Ventricle, Gland of neck removed at operation for case	62 "	Esophagus
13	55 "	2 "	Postcricoid	57 "	Esophagus
15	74 "	9 mos.	Pyriform sinus	74 "	Esophagus
19	37 "	2½ yrs.	Trachea	40 "	Metastasis in brain and lung
26	63 "	1 yr.	Pyriform sinus	63½ "	Neck

TABLE No. 6.

DEATHS FROM ANY CAUSE WITHIN THREE WEEKS OF OPERATION.

Case No.	Age at Operation	Number of Days Following Operation	Cause of Death
3	70 years	2 days	Diabetes, chronic nephritis
6	54 "	25 "	Abscess and gangrene lung
10	75 "	5 "	Pneumonia

I am not including in this series of cases laryngofissure, lateral or anterior translingual pharyngotomies, but simply the cases in which the larynx has been removed by single stage operation.

The number of patients operated on were thirty-two, thirty-one of whom were males and one female. The youngest patient operated on was 37 years, and the oldest 76 years in the males, and 55 years in the female.

The fourth decade of life, in my series, shows the greatest number operated on. Of these thirty-two patients operated on, eighteen are still living, the oldest eight years after operation and the youngest four months. Ten patients died from malignancy elsewhere than the larnyx, and it is worthy of note that all of these ten patients had extrinsic cancer, the time of recurrence in some being six months, in others two and one-half years, the average being one year and five months.

One patient on whom a laryngectomy was performed at the age of 63 years, died of apoplexy two and one-half years after operation without recurrence.

Of the thirty-two patients operated on, three died within twenty-five days of operation. One patient, age 70 years at time of operation, a bad operative risk to begin with, for he not only had malignancy of his larynx but diabetes as well as a chronic nephritis. This patient died two days following operation. Another patient, age 75 years at time of operation, developed pneumonia five days later. The other patient, age 54 years, on the twenty-fifth day following operation developed abscess and gangrene of the lung.

With the thirty-two patients operated on and three dying within twenty-five days of operation makes a mortality rate of 9⅜ per cent.

This does not compare with the brilliant results of other operators, but the patients operated on were not selected, many of whom were extrinsic in type.

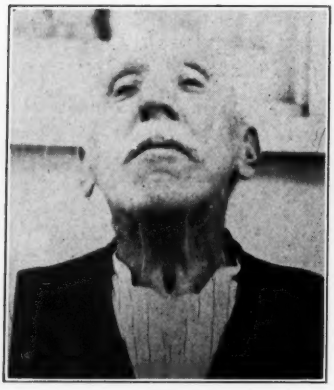

Fig. 6. Case No. 2890. G. W. O. Age at operation, 72 years. Date of operation, June 23, 1924.

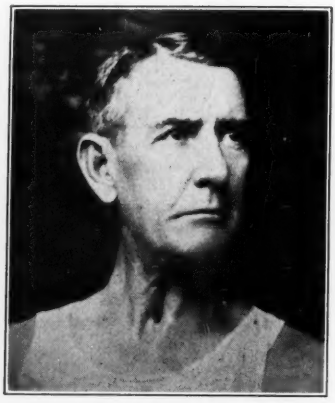

Fig. 7. Case No. 3538. R. C. McN. Age at operation, 50 years. Date of operation, August 4, 1925.

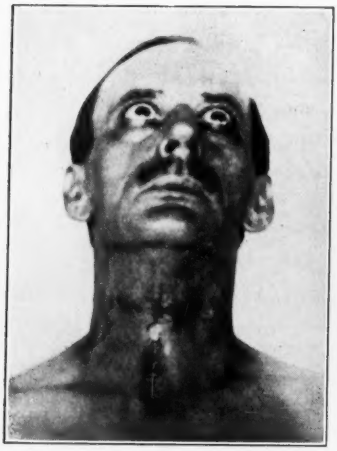

Fig. 8. Case No. 5272. J. O. Age at operation, 48 years. Date of operation, April 2, 1928.

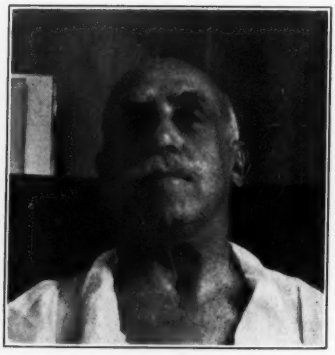

Fig. 9. Case No. 5663. L. B. Age at operation, 66 years. Date of operation, October 5, 1928.

Fig. 2.

Fig. 10. Case No. 5771. S. A. Age at operation, 49 years. Date of operation, November 22, 1928.

BIBLIOGRAPHY.

1. Some Principles in the Surgery of the Pharynx. Wilfred Trotter, M. S., London; LL. D. Edinburgh, F. R. S. Reprinted from The Lancet, October 17, 1931, p. 833.

2. Monograph on Cancer of the Larynx. John Edmund Mackenty, New York, N. Y.

3. Cancer of the Larynx. Sir St. Clair Thomson, M. D., F. R. C. S., F. R. C. P.; Lionel Colledge, M. B., F. R. C. S.; page 153, published by The MacMillan Company, New York, 1930.

REFERENCES.

An Analysis of 126 Cases of Malignant Disease of the Upper Air Passages Treated During a Period of Ten Years, 1921-1930. Lionel Colledge and Roydon Peacock, London. The Journal of Laryngology and Otology, March, 1932, Vol. XLVII, No. 3, page 161.

Malignant Disease of the Hypopharynx. Professor Von Eicken, Berlin. The Journal of Laryngology and Otology, April 1932, Vol. XLVII. No. 4, page 229.

Purvis Oration on The Surgery of Malignant Disease of the Pharynx. Wilfred Trotter, M. S., F. R. C. S. Reprinted from the British Medical Journal, February 13, 1926.

LXXX.

ATYPICAL FACIAL NEURALGIA, A SYNDROME OF VASCULAR PAIN.*

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PHILADELPHIA.

The term, atypical facial neuralgia, has become all inclusive for those types of pain about the face and head which do not fall into the classification of true trigeminal neuralgia. It has been more commonly accepted as the type of lower-half headache described by Sluder.

The present concepts of this syndrome are so varied, and the reports of successes and failures in treatment so numerous that confusion reigns on all sides as to its true nature and the proper solution.

In the hope of clarifying this subject, it has been taken up and studied from the neurological and anatomical aspects, observations being made possible by neurosurgical means of testing and destroying the suspected pathways of pain.

Our first consideration must be to establish or disprove the existence of this type of neuralgia. The patient usually complains of dull, aching, throbbing pain, situated deep in the eye and malar region, directed toward the ear. At times the pain may be referred to the back of the neck, behind the ear, and in the lateral aspect of the neck. Occasionally the pain radiates to the shoulder and into the axilla. (Fig. 2b.)

It would seem difficult at first glance to correlate such a wide distribution of pain into any single neurological mechanism, but we shall find it follows the arterial vascular tree of the neck and head, the branches of which merge and overlap and include the entire field under consideration.

There are those who still maintain that this pain syndrome complained of by so many patients is in reality a functional

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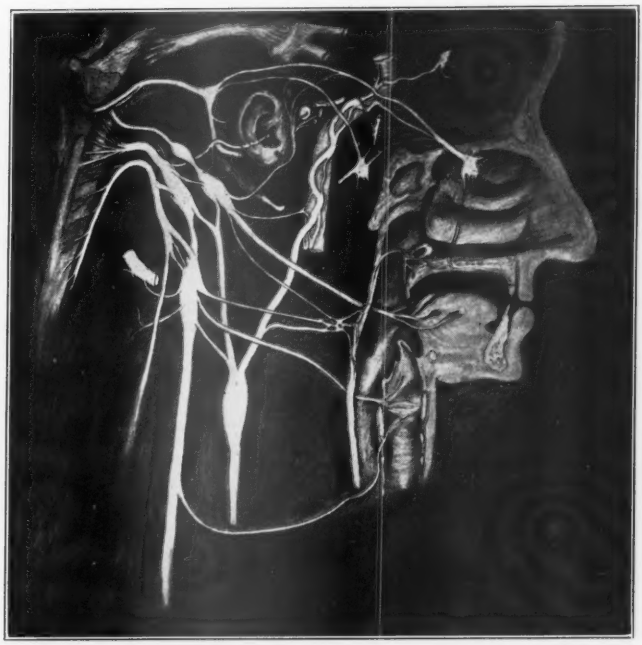

Fig. 1. Diagrammatic representation of the complex intercommunications between the vagus, glossopharyngeus and sympathetic branches, distributed to the head and neck.

condition, and they have placed the diagnosis "psychalgia" or "psychoneurosis" on those who wander from clinic to clinic to obtain relief.

Sherrington[20] has discussed the variable physiological reactions of pain responses, characterized by summation, collateral irradiation, slow culmination and slow subsidence. The effect of the psychic sphere, associations and emotional states does definitely enter the question.

Herrick[11] points out the close association in man between the primary thalamic reception zone for pain, and the cortical areas having to do with the intellectual and emotional life of the individual. The development in the organism of avoidance and pro-

tective reactions is phylogenetically very old, and intimately bound up with the visceral and peripheral responses.

Brouwer[1] [2] has shown the close relationship existing between the protecting pain mechanism and sympathetic vasomotor responses which occur reflexly in the pain arc. A close anatomical relation between the pain receptor cells in the dorsal gray matter of the cord, the short fasciculus proprius fibers attributed to pain function and the adjacent lateral cell column of the sympathetic system exists and has developed conjointly in the phylogenetic plan. This system of protective responses he has termed the "vital" mechanism, in contradistinction to more recent additions of informative sensory types carried in the posterior columns classified as "gnostic."

In the close interrelationship between pain and vasomotor responses we may well expect vascular changes which can be traced to definite arterial plans such as seen in angina, gastric colic and migraine. The direct implication of the pain fibers on the vessels themselves, as well as the change in the threshold of peripheral reception due to hyperemia, giving rise at times to involvement of the deep and superficial pain patterns, is frequently encountered.

If we admit that this large group of individuals suffer real pain and are truly worthy of our efforts toward its relief, then the next logical step is to determine the deep pathways of pain from the face, head and neck which are capable of carrying these sensations to the conscious brain.

It is quite evident that the large number of cases which refer pain in a vague way to certain definite areas of the face (Fig. 2b), especially deep in the region of the zygoma, deep in the eye, in the direction of the ear, deep in the ear and frequently down the neck, must have some common source of origin, and though the exact distribution or areas of pain may not be similar in each case, they will be found to be confined to more or less the same general area, and the difference lies in degree and extent of involvment, rather than dissimilarity in the types of pain.

Having closely scrutinized the structures capable of carrying this painful sensation, from a neurologic and anatomic viewpoint, we are confronted by a certain number of possibilities. The pain impulses, if peripheral in origin, must enter the central nervous

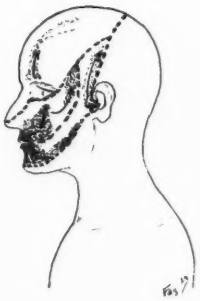

Fig. 2a. Distribution of pain in trigeminal neuralgia, sharp, flashing, shooting in character, with anatomical distribution to its branches.

Fig. 2b. Usual distribution of atypical neuralgia, deep, drawing, aching, throbbing pain in the malar region behind the eye, deep in the ear and down the neck, anatomical to the distribution of the vascular tree.

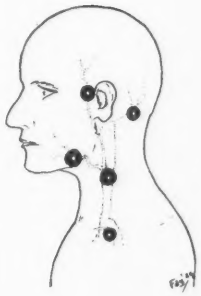

Fig. 2c. Points of tenderness to deep pressure over the vessels found in atypical neuralgia, not present in trigeminal neuralgia.

system by either the cranial or spinal nerve roots. By a process of elimination we may discard some of the cranial nerves in the light of our present knowledge. Certain other nerves and structures are open to question, their function never having been definitely established, especially in regard to pain.

This paper will deal with the further analysis and elimination of certain neurologic pathways which have been tested recently, in an attempt to solve the problem of pain presented by cases of metastatic carcinoma involving the floor of the mouth, the pharynx and the upper portion of the neck. From the information obtained by these operations (Table I), in which section of the trigeminus, glossopharyngeus, vagus, hypoglossus and sympathetic has been accomplished, it is possible to apply this knowledge in the hope of further elucidating the problem of atypical neuralgia.

It has now become generally accepted that the free nerve endings producing a netlike structure throughout the cutaneous tissues, and also in the muscles, along the bones, vessels and underlying the mucous membrane, are devoted to the function of the

appreciation of pain. This type of nerve ending is the only one seen in the cornea, the tympanic membrane and the dentine and pulp of the teeth, and pain is the only sensation that can be appreciated by these structures.

In 1914, Dejerine expressed the belief that pain was conducted by the "sympathetic" fibers contained in the cutaneous and muscular nerves. In 1915, Ranson,[17 18] in a series of brilliant experiments, showed that the unmyelinated fibers of the spinal nerves and dorsal roots were the true conductors of pain sense. As the sympathetic fibers are also unmyelinated, it is easy to see how confusion might exist between these two separate systems.

It is not necessary to suppose that the sympathetic system carries pain fibers, but these pain fibers, unmyelinated like their fellow sympathetic fibers, may follow a similar course and yet be distinct in many ways regarding function.

Herrick has pointed out that overstimulation may produce painful responses in other sensory groups. Undoubtedly, it is necessary to draw a sharp distinction between the terms used when alluding to sympathetic pathways and "sympathetic" pain.

It has now become accepted generally that pain fibers are unequally distributed throughout the spinal nerves and peripheral nerves, depending upon the importance of the part supplied regarding pain sensation. A general distribution of pain fibers throughout the musculature, the joints and over the vessels, as well as the linings of the mucous membranes, is present, but the mechanism, aside from trauma or inflammation, which acts to bring them into function, has frequently escaped detection.

The histologic presence of both myelinated and unmyelinated fibers has been demonstrated, on the adventitia of vessels of the brain, by Hassin.[10] The physiologic function of the sympathetic fibers to the cerebral vessels has been established by Cobb[3] and Forbes and Wolff,[8] who have observed, through a glass window in the skull, definite cerebral vascular responses to cervical sympathetic stimulation, and these changes have been recorded by microphotographic methods.

Neurosurgeons have frequently observed, when exposing the brain (with the patient under local anesthesia), that there is no perception of pain to irritation or stimulation of the dura if the

large branches of the middle meningeal artery are avoided. Electrical stimulation, pinching or trauma to these vessels brings forth a sharp cry of pain which may be localized in a general way to the side of the head on which the stimulation occurs. If, however, the main branch of the middle meningeal artery is tested or clamped, severe pain is at once referred deep in the eye.

One may stimulate, section, or cauterize the cortical surfaces of the brain without producing a sensation recognized by the patient as pain. Frequently paresthesias may be produced from stimulation of the post-Rolandic area, but these are tingling and burning in character. The large branches of the middle cerebral artery, if subjected to stimulation or irritation, bring forth a definite response of pain, usually localized deep in the temporal region and behind the eyes.

These clinical observations would seem to indicate that certain of the fibers, demonstrated histologically by Hassin, have a definite pain function. The existence of sympathetic responses, noted by Cobb, Forbes and Wolff, indicates that others are probably related to the sympathetic and parasympathetic systems.

It has become necessary to differentiate the many types of pain encountered in the head and neck. Halphen, Monbrun and Tournay[9] have recently made such a classification of the cephaleas. Neurologically, we distinguish between superficial pain and deep pressure pain, but the difference between these two phenomena cannot be recognized by any distinction in the type of nerve endings or fibers supplying the superficial areas and those which supply the deeper structures. There is only a difference in number and degree of associating groups, the deeper structures being supplied by fewer free nerve endings. Nevertheless, clinically there is no doubt as to the difference between the character of pain produced from an injury such as a superficial abrasion of the skin, into which salt or an irritant has entered, as compared to the dull, throbbing pain encountered in headaches or pressure pain from a bone tumor or an injury to the deep muscle layers.

The difference between the type of pain complained of in atypical neuralgia and that seen in the true type of trigeminal neuralgia partakes of these distinctions; the latter is associated with sharp, shooting flashes of burning, electrical pain, the former

TABLE I.

SECTION OR ABLATION.

Case	V.	VII.	IX.	X.	XII.	C2. C3.
1. M. W.	Total section of root					
2. Eliz. C.		Paralysis following mastoidectomy				
3. E. S.	Avulsion 2nd, Injection 3rd				Peripheral section	
4. J. F.						Section of roots 2-3-4. Rhizotomy
5. C. W.						Section of roots 2-3-4. Rhizotomy
6. J. J.	Total section of root					
7. C. D.	Total section of root	Peripheral section	Intracranial section	Intracranial subtotal section of root		
8. C. K.	Total section of root					Superficial cervical plexus
9. E. B.			Extracranial branches	Extracranial sensory branches	Peripheral section	Avulsion, superficial cervical plexus

10. J. S.	Section 2nd and 3rd division			Extracranial sensory branches	Peripheral section	
11. E. T.	Total section of root		Extracranial and jugular branch sympathectomy	Extracranial sensory branches. Laryn-pharyn.	Peripheral section	
12. E. C.	Sub-total		Extracranial section	Auricular branch and sensory branches	Branches to vagus. Peripheral section	Branches of plexus sectioned
13. F. F.				Peripheral sensory branches		Superficial branches
14. E. B.	Total section of root		Intracranial section			Section of roots, 2-3 Rhizotomy
15. F. R.	Total section of root		Peripheral section	Peripheral sensory branches	Branches to vagus	Avulsion of occip. Maj. Section of superficial plexus
16. A. De L.				Peripheral section of sensory branches	Branches to vagus	Superficial branches
17. J. C.	Subtotal section of root			Total section, intracranial root		
18. M. R.				Peripheral sensory branches	Peripheral section	
19. R. K.	Total section of root	Upper branches cut	Extracranial section	Extracranial sensory branches	Peripheral section	Section of superficial branches

TABLE I (Continued).

SECTION OR ABLATION.

Case	Spheno-Palatine	Superior cervical ganglion	Cervical sympathetic chain	Common carotid artery	Bifurcation, external internal carotid	Result
1. M. W.						Carotid and vascular pain remained.
2. Eliz. C.						Unimproved.
3. E. S.						Return of pain, unimproved.
4. J. F.						Carotidynia and vascular pain remained.
5. C. W.						Carotid and vascular pain remained.
6. J. J.		Section of cephalic branches	Middle and inferior ganglion avulsed	Denudation		Unimproved.
7. C. D.						Improved of superficial pain, but carotidynia and vascular pain remain.
8. C. K.						Carotid and vascular pain unimproved.
9. E. B.		Branches to vagus	Removal below superior ganglion			Great relief of pain.

10. J. S.						Greatly improved.
11. E. T.		Branches to vagus. Jugular branch				Slight improvement. (Died after intracranial and IX).
12. E. C.				Branches to vagus		Complete relief of pain.
13. F. F.					Denudation	Greatly improved.
14. E. B.						Improved of superficial pain, but carotidynia remained.
15. F. R.		Ablation	Ablation	Partial stripping	External carotid found obliterated	Unimproved.
16. A. De L.		Resection	Ablation	Stripping	Denudation	Improved, almost complete relief.
17. J. C.						Complete relief of pain and carotidynia.
18. M. R.		Resection				Improved.
19. R. K.	Resection	Removal	Ablation	Stripping	Denudation	Complete relief of carotidynia and vascular pain.

with deep, dull, aching, boring pain, often throbbing in character, which can leave no doubt as to its characteristics or diffuse distribution. In either case, if the lesion be peripheral, the fibers must find their way in some manner through the cranial or spinal nerves and thence to the brain and conscious levels, before pain can be appreciated..

In dealing with pain and its relief by surgical section of certain vascular pathways as evidence in favor of establishing the course and direction of such fibers, it is not enough to obtain simply a clinical freedom from the pain on the part of the patient. An anesthesia must also be demonstrable.

Pain perception and recognition may be greatly influenced by vasomotor changes in the periphery, such as congestion and relative ischemia. The relief of pain obtained by various methods may be dependent upon altered vasomotor properties, which, when corrected or influenced by surgical section of large vascular fiber groups, brings freedom from the symptom designated as pain but in no way establish the fact that actual pain pathways have been severed.

I am impressed with the fact that often relief of pain, in atypical neuralgia, is not immediate, after ablation of the vascular, sympathetic and vagus sensory fibers. The improvement may be partial at first, with a gradual relief which amounts to almost complete subsidence of pain at times. This implies a period of adjustment and must be considered as a secondary factor in the pain mechanism.

The threshold for pain perception is greatly enhanced when hyperemia is induced (immersion of the extremity in hot water for a short period), and is greatly reduced when ischemia is present (exposure to cold or immersion in cold water for a period sufficient to maintain vasoconstriction). There has been no alteration or "pathologic" change in the end organs or pathways demonstrable under these conditions, but the element of pain, as perceived by the patient, has been greatly altered.

In dealing with vascular pain, we are necessarily forced to consider both factors. The threshold for pain perception must include the component responsible for the hyperemia, as well as the pain end organ, both of which are closely interrelated and

associated with the central nervous system and the brain. The "vital" protective pain mechanism and vascular sympathetic response are frequently reciprocal and augment each other. Thus a change in vascularity to a part may influence the pain threshold in the area involved.

It has been, therefore, impossible to separate these two components with any degree of certainty. In the experimental animal, one cannot determine "anesthesia" or "pain" with any degree of certainty. What the subjective responses to appropriate stimuli are can only be a matter of surmise from the degree of response noted. In man the possibility of making repeated observations on the pain sensitivity of the vessels, after denudation and section of the fibers distributed to them is limited, because of their deep location.

METHOD OF VASCULAR PAIN DETERMINATION.

Testing the arterial branches by means of a fine hypodermic needle connected to a faradic current, after careful injection of novocain about the skin and superficial layers, has been one means of determination in this series. Pressure of the artery against a fixed bony prominence is fairly satisfactory. In testing for vascular sensitivity, care must be exercised to avoid points associated with known sensory nerve trunks. Direct stimulation of the exposed arterial sheath, the connecting vagus fibers and arterial branches was undertaken repeatedly, in operations upon the neck. In several cases, re-exploration after a previous carotid denudation has offered a means of comparison between the responses originally present and those found some months after stripping of the vascular sheath.

In many of the cases, direct pressure on the arterial tree or faradic stimulation of the vessel wall through the skin was made possible, without interference of the overlying sensory fields, because the trigeminal and posterior cervical roots had previously been destroyed, for the relief of pain produced by malignant and invasive growths of the face, head and neck.

In spite of total superficial anesthesia demonstrable over the face and neck, in such combinations as destruction of the trigeminus, glossopharyngeus and upper cervical roots, there remained

frequently dull, throbbing pain referred to the field of the large vascular branches, and tenderness to pressure as well as pain on direct stimulation could be obtained in the facial, temporal, occipital and carotid arteries. (Fig. 3.)

OBSERVATIONS ON VASCULAR PAIN.

These tests, carried out on patients with large areas of anesthesia, clearly suggested the existence of an underlying pain mechanism, the fibers of which followed the vascular branches and network and remained independent of the craniocervical sensory nerves.

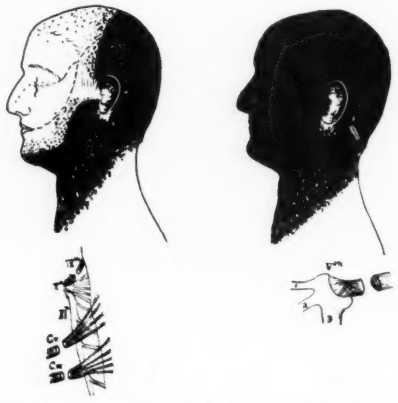

Fig. 3. Widespread area of superficial anesthesia in case E. B. produced over the head and neck by section of the trigeminus, glossopharyngeus and upper two posterior cervical roots. Vascular tenderness and deep pressure pain over the vessels of the neck and head remained.

The question as to the point of entry of these vascular pain fibers into the central nervous system has claimed our attention for the past five years. By a gradual process of elimination, using the Sherrington block method of widespread anesthesia, where malignancy justified the extensive root destruction, it has been possible to determine what cranial nerves do not carry these fibers, as well as to indicate by the negative results obtained by various combinations of sensory nerve section the probable structures implicated, as far as vascular pain is concerned.

A study of Table I indicates that the vagus nerve is the only cranial nerve whose destruction influenced the clinical course of the vascular pain, and yet the cases in which this structure was sectioned intracranially presented complications which have failed to establish a definite loss of sensitivity of the vessels.

Intracranial vagus root section, combined with trigeminal root section, was accomplished in one case (J. C.). Although symptomatic relief was complete, the field of anesthesia was not sufficient to establish the pain values which could be attributed to this nerve. In my opinion, there seems little doubt as to the influence the vagus has upon this syndrome of pain, as other cases of extracranial division of its sensory branches have also given symptomatic improvement, but one cannot overlook the possible vasomotor release which may underlie the pain mechanism, and not indicate that pain fibers actually return to the brain through the vagus.

In order to effect a loss of vascular tenderness and "carotidynia" it has been necessary to denude the common carotid artery, especially its area about the bifurcation, and to sever branches to the vagus as well as the jugular branch of the superior cervical ganglion.

Stimulation by faradic current about the bifurcation of the carotid is extremely painful to the patient. The sheath of the vagus, composed of fine branches from the carotid, is also extremely sensitive. Pain may be referred into the tongue, upper and lower jaws, the face, about the orbit, temporal region, occipital area, deep in the eye and head, and into the throat by appropriate selection of points about the arterial bifurcation. (Fig. 4).

In two instances (F. F. and M. R.) a large gland was disclosed tightly imbedded in the carotid bifurcation crotch, bound down by fibers from the sheath, the manipulation and irritation of which referred extreme pain to the eye, upper jaw and temporal region.

After freeing this structure and its removal in the case of M. R., the external carotid artery at its source was seen to be greatly compressed and almost obstructed, an hour-glass constriction persisting that was not of the spasmodic type, but clearly a pressure obliteration.

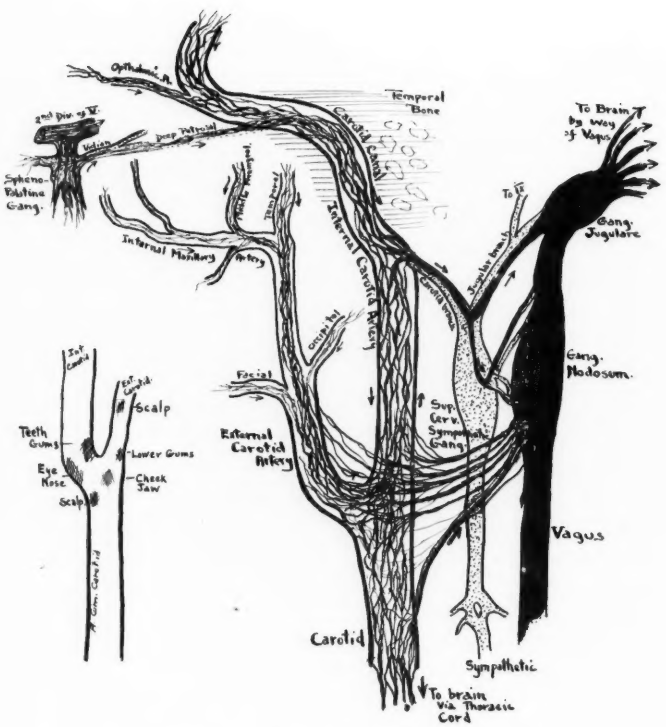

Fig. 4. Right common carotid artery and bifurcation with areas stimulated by faradic current in case F. R. showing reference of pain as determined by the patient during the period of stimulation.

Fig. 5. Schematic representation of the vascular pain fibers and their probable points of entry into the vagus. Some pathways undoubtedly descend to enter the thoracic cord.

The pain references which have been noted when stimulation was applied to the individual vascular branches will be found in Table II.

Destruction of the cervical sympathetic chain alone, below the superior cervical ganglion, does not destroy sensitivity of the vessels or obliterate the vascular type of pain. Common carotid

TABLE II.

Arterial Branches Stimulated	Pain Reference
1. Meningea Media	Deep in the eye. Temporal region; deep.
2. Cerebri Media	Deep in the eye. Headache.
3. Temporalis Superficialis	Ear. Scalp temporal region.
4. Maxillaris Interna	Eye. Malar region.
5. Occipitalis	Scalp occipital region. Back of neck.
6. Maxillaris Externa	Inner canthus of eye. Cheek.
7. Lingualis	Tongue. Tonsillar region.
8. Carotis Externa	Scalp, malar region. Gums, teeth, jaws.
9. Carotis Interna	Deep in ear. Deep in eye. Headache.
10. Bifurcation	Scalp, ear, nose, teeth. Gums, cheek, jaws, tongue.

denudation alone, when carefully undertaken, does give decreased sensitivity of the vessels for pain and symptomatic relief. The results have been extremely variable and, in my experience, incomplete.

Proper removal of the carotid sheath is not a simple or an easy undertaking. Many surgeons have been content to open the sheath, dividing the fibers which present anteriorly, but they usually fail to deal adequately with the posterior portion of the sheath. Fear of rupture, difficulty in rotation and proximity to the vagus, as well as perivascular bleeding, frequently have led to only partial sympathectomies, grossly incomplete and of insufficient depth. Operators have repeatedly reported negative results from their efforts and have attempted to discredit its possibilities, based on such imperfect technic. These reports have only con-

fused the issue. A complete and satisfactory carotid denudation must include the bifurcation and the two large branches arising from it to be effectual. It is a long, difficult and delicate procedure, the results to be obtained being dependent upon the thoroughness of the operator.

NEGATIVE DEDUCTIONS.

Atypical neuralgia, which we have now under consideration, does not yield to section of the posterior root of the trigeminus. Deep, aching pain in the face may remain after section of the posterior root. Tenderness to deep pressure may be demonstrated along the vessels in the area of anesthesia following destruction of the fifth nerve.

As the pain of atypical neuralgia persists after section of the fifth nerve, and painful spots may be demonstrated in the area of anesthesia by deep pressure on the vessels, we can at once rule out the possibility of its entering through the fifth cranial nerve.

As for the other cranial nerves, we can, of course, disregard the fourth, sixth and eighth, as the function of these nerves is well known. We must consider the possibilities offered by the seventh, ninth, tenth, eleventh and twelfth, as well as the cervical supply which overlaps the head and face. The sympathetic system with branches from the superior cervical ganglion, which supplies fibers directly to the large vessels of the head and neck, distributed to the very parts involved, must entail special consideration.

Davis[4] has shown that deep pressure pain sense, in the cat, may be carried by the seventh nerve, a clinical observation made by Spiller,[21] in 1907, but experimental evidence has failed to show that the seventh nerve, peripheral to the geniculate ganglion, carries other forms of sensation.

Ramsay Hunt,[12 13 14] describes a form of herpes zoster associated with facial paralysis, and ascribes a pain function as well as an area of sensation to this nerve.

It has been shown[6] that this area ascribed to the seventh closely corresponds to that known to be supplied by the vagus, and that branches of the vagus usually accompany the facial nerve to its

point of exit from the facial canal, so that much doubt exists as to the justification of attributing common superficial sensation to this nerve.

It may be possible that in Hunt's cases the inflammation responsible for the facial paralysis and herpes included both structures. We have no experimental or clinical evidence which would bear out the consideration of the seventh nerve as a possible pathway for entry of residual pain that persists after destruction of the fifth. In two cases included in this paper, destruction of the seventh as well as the fifth did not obliterate the vascular tenderness or the pain referred to the face.

In a similar manner, it has been possible to establish that the ninth nerve does not carry deep pain sensation from the face through its intracranial roots. Its function, at least intracranially, apparently is devoted to the special sense of taste and to the vasomotor responses which are secondary to the gustatory reflex. There is considerable evidence to show that the intracranial roots of the eleventh and twelfth are, in all probability, purely motor. This then leaves us with three nerve pathway possibilities.

If pain is perceived along the vascular tree, in cases of atypical neuralgia, it must enter the central nervous system by one or more of three routes:

1. The vagus,
2. The sympathetic chain, or
3. The carotid sheath to the base of the neck, and thence into the cervical-thoracic posterior roots.

In my[6] case of carcinoma of the mouth, in which the posterior root of the fifth was sectioned, the intracranial root of the ninth destroyed, and the upper two (C_2 and C_3) posterior, cervical roots obliterated, the patient still suffered from vascular tenderness and pain in the ear, and a deep, throbbing, aching pain persisted in the area of demonstrated anesthesia about the face and eye. The cutaneous tests showed anesthesia of the entire side of the face and upper neck with the exception of a small area within the concha of the ear which was attributed to the vagus. This case, after close observation for a period of fifteen months, has finally come to autopsy, and it was possible to demonstrate the complete destruction of the nerves attempted.

As the patient was not entirely relieved from the pain in her ear or face, and as it could be demonstrated that pressure over the vessels of the face and head, on the side of anesthesia, was capable of producing pain, it leaves a fairly definite assumption that these pain fibers must reach consciousness below the fourth cervical root or through one or two structures, or both, the vagus or the cervical and sympathetic carotid sheath ramifications.

Our problem has been confined recently, therefore, to these structures. In order to determine the pathways more definitely several combinations of nerve section have been undertaken. It was found in one case, where the intracranial root of the vagus was sectioned, that all pain in the ear, and pain referred to the throat were relieved, as well as tenderness to deep pressure along the upper aspect of the vessels of the head and neck.

In another case, in which the pain was acute, situated behind the ear and down the lateral aspect of the neck, being frequently referred into the tonsillar region, produced by pressure on the lateral aspect of the neck at the upper level of the hyoid bone, an exploration of this area of tenderness to pressure was made. The hypoglossal nerve was encountered at this operation. As the patient was under local anesthesia, it was possible to demonstrate the function of the hypoglossal nerve. Stimulation by faradic current of the hypoglossal nerve produced pain referred to the tonsillar region, the base of the tongue and deep in the ear. Novocain injected into the nerve relieved the pain produced by stimulation. Injection of alcohol increased it. Section of the nerve produced a relief of this type of pain, as well as the spasms which were associated with it.

The hypoglossal nerve is essentially motor, but receives a definite filament from the vagus. In two other cases, separation of the vagohypoglossal connections present at the crossing of these two structures in the neck, gave relief from painful stimulation of the hypoglossal trunk. Stimulation or traction on the vagus stump gave direct reference of pain into the tongue, pharynx and ear. These observations taken with the case of intracranial vagus root section, indicate that the hypoglossal nerve carries pain fibers which it receives extracranially directly from the vagus.

During the extracranial section of the sensory nerve branches of the vagus, in a case of carcinoma, this same phenomenon was demonstrated. Traction upon the hypoglossal nerve produced pain in the ear and referred to the tonsillar area. The exposure included the sympathetic, cervical, superior ganglion, the vagus, the glossopharyngeus and the hypoglossus. It was possible to test these structures with the patient under local anesthesia.

It was found that there were many connections in a network between the vagus and the superior cervical ganglion; that section of this network connecting these two structures was associated with pain referred to the ear, and stimulation and traction produced a response on the part of the patient which left no doubt as to the remaining pathways of pain. The cervical sympathetic had been sectioned below the ganglion before the stimulation was undertaken. It seems fairly evident, therefore, that no matter what the course of these pain fibers is, in relation to the sympathetic system or its branches, they are probably in close relation to the vagus in its connections with the superior cervical ganglion and that the deep pain pathways referred to the structures in the region of the ear, tongue and pharynx, are, in all probability, through the vagus, as the sympathetic chain had been interrupted below the superior cervical ganglion. Those impulses still reaching consciousness had only one remaining cranial nerve point of entry, i. e., the vagus.

Attention was later centered upon the structures of the neck as a pathway for pain referred to the face, along the sympathetic system, especially the cervical sympathetic and carotid sheath, as a conveyor of pain fibers, and the vagus as a possible source of entry of these pain fibers to the brain. The attempted operative combinations, applied in an effort to explain the various pathways of atypical neuralgia as they might conform to a neurologic or vascular mechanism, have become somewhat clarified through these observations.

VASCULAR PAIN.

In 1927, I[5] called attention to the presence of tenderness to pressure on the carotid arteries of the neck, in cases of atypical neuralgia.

If the thumbs are placed on the common carotid arteries, just below the bifurcation, and the structures pressed back against the transverse cervical processes with a rolling movement, a severe reaction of pain is produced on the side of the atypical neuralgia. This response I have termed "carotidynia," and it has been found present in almost every case where patients have complained of chronic, dull, aching pain referred to the eye, deep in the malar region and traced back to the ear, behind the ear or down the neck.

Associated with this sign is a reflex dilatation of the pupil which occurs in the normal, but in cases where atypical neuralgia was well established, the pupil on the side of the tenderness (carotidynia) would dilate more fully and remain dilated longer than its fellow following such carotid manipulations. This delayed anisocoria is quite striking, and without knowledge as to the side of the pain, the examiner may determine this fact by the above tests before the patient has disclosed the side involved.

In several cases of pansinusitis, carotidynia was not only extreme, but pressure on the vessel in the neck referred pain directly to the eye, the malar region and the upper jaw. Although I have repeatedly obtained reference of pain into the lower half of the face by carotid irritation, in acute sinus and mastoid infections where atypical neuralgia was present, yet I have only seen one case in the chronic groups with long histories of this type of pain, where carotid manipulation brought forth direct and immediate reference of pain, deep in the eye and malar region. Many patients have complained of these manipulations increasing or bringing forth an attack, but aside from the carotidynia, no projection of pain forward was appreciated at the time. "Carotidynia" has not been found to be present in any case of true trigeminal neuralgia.

As the manipulation of the artery just below the bifurcation responsible for the "carotidynia" must also involve the vagus and adjacent cervical sympathetic, it was necessary to determine what part these structures played in the production of the pain.

The part that the vagus plays in this pain has already been discussed. The influences of the sympathetic chain and the carotid sheath remain undetermined.

The observations on R. K. (Case 19), over a period of three years, established several factors of importance. The patient gave the usual history of deep, dull pain over the left side of the face, confined to the eye, malar region, and directed back to the ear, at times extending down the neck. The pain was worse at night and became unbearable. He had had many sinus operations, much nasal and dental surgery, to no avail. Fruitless injections of the second division added an intolerable paresthesia of trigeminal character to the original syndrome. When seen by me in 1926, he presented atypical neuralgia, on top of which had been introduced a traumatic trigeminal neuralgia. As the pain prevented sleep, he insisted on relief, and the following operative procedures were undertaken during the ensuing three years:

1. Section of the trigeminal root (Spiller operation), with relief of the trigeminal pain and paresthesia but no relief of the atypical neuralgia.

2. Resection of the sphenopalatine ganglion (transzygomatic approach, as injections are uncertain and unreliable) and upper branches of the facial. No relief of the symptom complex.

3. Ablation of the cervical sympathetics from below the superior cervical ganglion. (Hunter-Royle operation.) No relief.

4. Stripping of the common carotid artery. Great relief, though not complete (40 per cent improvement). Pain still deep in the ear and malar region.

5. Section of the hypoglossus. Relief of deep pain referred to the angle of the jaw and region of the ear.

6. Extracranial section of the glossopharyngeus (Adson operation). Section of the sensory branches of the vagus, jugular branch to the vagus and Arnold's nerve. Complete relief of pain in the malar region.

7. Division of vagus sheath, division of vagus fibers to the internal and external carotid arteries, denudation at the bifurcation. Marked relief of "atypical neuralgia" pain.

An analysis of this case has shown that "carotidynia" persisted after ablation of the sympathetic chain and sensory branches of the vagus. Stimulation of the vessels at the seventh operation disclosed the fibers at the bifurcation entering the vagus sheath, referred pain to the malar region and the face, especially when

the facial arterial branch was tested, as well as the external carotid. The internal carotid gave pain deep in the parietal area.

"Carotidynia" seems definitely to be of vascular origin, as it remained until the seventh operation. At the present time the patient has no "carotidynia."

M. R. complained of intense pain in the eye, referred back to the ear and down the neck, on the right side. Supra-orbital neuralgia was also present. On the insistence of her physician, the trigeminal root was sectioned, without relief of her pain.

The following operations for relief of the atypical neuralgia ensued:

1. Ablation of the cervical sympathetics.
2. Extracranial section of the sensory vagus, glossopharyngeus and Arnold's nerve.
3. Avulsion of the occipitalis major.
5. Stripping of the common carotid artery, below the bifurcation, and section of the superficial cervical plexus.

The patient has had return of pain after each operation, and although somewhat improved, still has a great deal of pain in the malar region and the eye.

In this case the vagus sheath and fibers around the bifurcation were not removed and carotidynia still remains. An enlarged gland at the bifurcation was found, causing almost complete occlusion of the external carotid artery. The pathology prevented carrying the denudation higher.

Carotidynia remained even after section of the sensory branches of the vagus and cervical sympathetics, as well as denudation of the common carotid sheath, indicating the unsectioned fibers at and above the bifurcation, as well as the vagus sheath must still contain the pain arc reaching consciousness cephalad to this point (i. e., through the vagus).

Since the studies on sensation of the head and neck presented before this society, in 1926, by Spiller and myself, vascular pain has been tested after a large series of combined nerve sections, for relief of pain in malignancy or chronic neuralgias, so as to determine the pathways of entry into the central nervous system of those pain fibers demonstrable clinically associated with the cerebral and cervical vascular tree.

The various combinations of nerve sections given in Table I have led to the distinct impression that the vascular pain fibers of the head and face find cranial nerve entry into the vagus, for the most part, near the bifurcation of the carotid. A considerable portion of the fibers pass down along the vascular structures of the neck to find entry into the lower cervical and upper thoracic cord. (Fig. 5.)

Further confirmation that a large portion of these fibers from the deep vascular tree of the face enter into the upper thoracic and lower cervical cord has been obtained by inducing spinal anesthesia and observing the objective and subjective responses during acute attacks of atypical facial neuralgia.

The control of spinal anesthesia has been carried out by placing the patient on the operating table and tilting the body to an angle of thirty degrees with the head down. A spinal needle is introduced into the lumbar sac, and another needle introduced into the cisterna magna. The cisternal needle is connected by means of a rubber tube to a graduated burette containing 50 cc. of saline solution. The burette is raised until the level of the saline in the burette is slightly higher than the spinal canal in the lumbar region, and spinal fluid is forced out of the lumbar needle. The burette is then lowered so that the level of the saline falls below the head, and spinal fluid flows rapidly into the burette, drawing air back into the lumbar sac through the lumbar needle, as the column of fluid descends along the spinal cord. One cc. of the ascending type of spinal anesthesia is introduced into the lumbar needle and drawn down the spinal canal to any desired level.

As the fluid in the spinal canal seeks the same level as the solution in the burette, it is evident that the spinal anesthesia may thus be drawn down along the spinal cord and stopped at any level desired. A reverse flow may be instituted by raising the burette and forcing the saline and cisternal fluid back into the cisterna magna (Fig. 6), washing out the spinal anesthesia through the lumbar needle when the test has been completed.

In this way it is possible to produce a spinal anesthesia to any level and to note by the subjective and objective tests the exact segments of the spinal cord anesthetized.

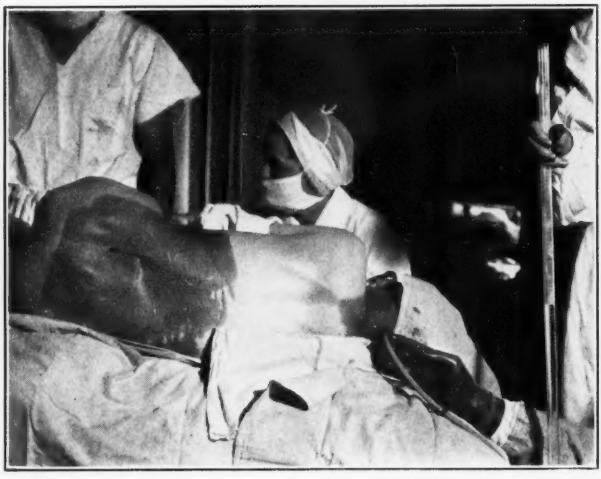

Fig. 6. Position of the patient for controlled spinal anesthesia. Note cisternal needle connected with burette; the level of fluid in the burette slightly higher than lumbar needle. Fluid flows into cisterna magna and out of lumbar needle.

In atypical facial neuralgia the pain distributed to the malar region and the tenderness to carotid pressure disappear immediately when the anesthesia reaches the first thoracic segment of the cord, as demonstrated by loss of pain sense in the axilla. Subjectively the patient feels numbness in the little finger and the ulnar aspect of the hand. (Fig. 7.)

The deductions obtained from these tests indicate that the pain fibers from the face follow the carotid arteries, the internal and external jugular veins and other vascular structures, and make their entry into the spinal cord in the upper three thoracic roots. Chordotomy below this level has failed to produce a loss of this type of pain and suggests that the point of entry is at least above the fifth thoracic segment.

In view of these findings, it is understandable how second thoracic ganglionectomy and the operations on the inferior cervical and upper thoracic sympathetic chain have been associated with certain degrees of relief, such as have been reported.

Fig. 7. Burette lowered after introduction of spinal anesthesia into lumbar needle. Spinal anesthesia gradually drawn down along the spinal cord as fluid flows from the cisterna magna into the burette. Note level of fluid in burette establishes exact level of fluid in spinal cord. Operator may arrest anesthesia at any point, and following desired anesthesia, may wash out the spinal canal by raising the burette to a level above the lumbar needle.

The futility of attempting to interrupt all of the vascular pain pathways from the head is clearly indicated in the large number of operations attempting to combine section of the nerves and the vascular pathways, such as are presented in table I, covering a period of eight years in search for a point of entry of these deep vascular fibers.

That the vagus may play a part, either through its vasomotor influence or directly, seems clearly indicated in the series reported, but that the combination of section in certain cases may have been more fortunate than others cannot be overlooked, as the results are not in complete accord. The emphasis to be placed upon the fibers which descend into the thoracic cage has not been recognized until recently.

Faradic stimulation of the internal and external jugular veins, as well as the vertebral and carotid distributions, under local

anesthesia, clearly indicates that these structures are well supplied with pain fibers, frequently referring their pain directly into the base of the head, the neck, deep in the ear, the malar region of the face, the base of the zygoma and into the teeth and gums.

These observations indicate that the interruption of the pain pathways by means of a high cervical chordotomy on the opposite side may be justified in extreme cases, as the pain fibers which enter the spinal cord in the upper thoracic area cross immediately to the other side and ascend in the anterolateral column, which has been amenable to surgery by the Spiller operation, and proven of great value in cases of involvement of the lower one-half of the body.

That high extensive cervical chordotomy is possible has been demonstrated recently by Putnam (for relief of dystonia). The anterolateral columns of the cord have been divided by me at the fifth cervical segment in one case of extreme atypical neuralgia, with complete relief of pain referred to the face, jaw and malar region.

Foerster[7] has also pointed out, recently, these vascular pain fibers on the carotids and its branches, and indicates their entry into the spinal cord as low as the fifth thoracic root. His studies did not include the relationship of these fibers to the vagus.

SOME FACTORS IN THE ORIGIN OF ATYPICAL NEURALGIA.

A. *Infection and Inflammation.*

Sear's work[9] has been most enlightening as a possible means of explaining why, in a certain group of cases, we might expect the phenomenon known as atypical neuralgia. Those who are actively engaged in the observations of acute middle ear and mastoid infections frequently see extensions of the inflammation in certain cases beyond the confines of the mastoid cells and the temporal bone (such as we frequently see in brain abscesses of mastoid origin, producing a collection of pus which has eroded through the upper aspect of the temporal bone and has become extradural or even pierced the dura to enter the hemisphere). There is likewise the possibility of extension of the disease below the mastoid or into the carotid canal, where the vascular pain

fibers and anatomic continuity would give adequate explanation for not only Gradenigo's syndrome but also the production of certain of the atypical neuralgias.

It is important to consider the possible consequences of an inflammation which has extended beyond the mastoid cells and has reached the confines of the carotid canal, such as described by Wilkinson.[23] If the inflammation reaches the sheath of this artery it will involve the pathways of the sympathetic on the artery, passing forward to control the ocular sympathetic supply which probably also conducts with their fibers sensory additions from the vagus, producing in the irritative stage a slight dilatation of the pupil. If the pain fibers associated with the sympathetic are present, supplying the structure of the eye involved, we should expect pain referred deep in the eye, along with the ocular sympathetic phenomena of anisocoria.

If purulent infection should reach the carotid canal and invade the carotoid sheath it would have two possible directions of extension. One, anteriorly and superiorly, to follow the artery to the region of the Gasserian ganglion, which overlies it laterally, and on into the cavernous sinus, which partially envelops the artery as it passes forward toward the anterior clinoid process. At this point the sixth nerve lies close to the carotid sheath in the cavernous sinus, and it is frequently observed that the so-called Gradenigo's syndrome is associated with extreme pain in the face (involvement of the Gasserian ganglion), accompanied by sixth nerve paralysis and occasionally by cavernous sinus thrombosis.

On the other hand, if the inflammation passes downward along the carotid sheath, it must emerge in the lower aspect of the canal, close beside the vagus and the superior cervical ganglion, involving the carotid branch from the cervical sympathetic, as well as the tissue plane in which these structures lie, close beside the carotid artery. Another explanation might be offered, in that inflammation associated with mastoid disease or middle ear disease may also involve the structures internal to the mastoid and sternocleidomastoid muscle. If this fascial plane be involved, it would produce adhesions around the branches of the vagus and the superior cervical ganglion, so that both of these structures might come to be involved in a chronic process, which could

refer pain later by irritation in the direction of the fibers. That is, the lesion being situated below the mastoid and involving the fibers which pass forward into the vessels, could refer the pain to the malar and retro-orbital region. In so doing it could be interpreted for antrum pain, leading to a mistaken diagnosis as to the structure involved.

In two autopsies I have traced a cavernous sinus abscess to direct extension from the mastoid by way of the carotid canal and sheath.

The frequency of middle ear disease in childhood, as well as the many types of nonproductive sinus and mastoid infections, devoid of pus but characterized by intense congestion and inflammation, gives ample opportunity for such processes to occur, and recover, without surgical intervention. The frequent enlargement of the deep cervical lymphatic glands is evidence of the direction of drainage and inflammation from sinus and tonsillar infections.

The reactions to tonsillar infection and retrotonsillar abscess, evidenced by glandular swelling in the neck, make possible inflammatory adhesions in the deep fascial planes containing the vagus and carotid sheaths. Such adhesions and enlarged glands have been encountered in many of the cases operated upon, the vessels and vagus being closely adherent in a firm fusion of the structures. The tonsillar fossa is separated by only 1 to 2 cm. of tissue from the internal carotid artery and sheath, presenting the possibility of inflammatory extension into the deep structures of the neck.

B. *Reflex.*

Reflex vasomotor disturbances noted in the face and neck from visceral diseases are well recognized. Lapinsky[15] has described the responses even to pelvic diseases. Blushing, menopausal flashes and gastro-intestinal migraine are frequent clinical examples.

Four cases of atypical facial neuralgia, in my series, confined to the right side proved to have their origin in gall-bladder disease, operative correction on the gall-bladder relieving the atypical neuralgia permanently. Several cases have shown lesions in the apices of the lung. Here the extensive pathology involved the structures at the base of the neck and the vascular tree to the face. Frequently, these cases have been victims of extensive nasal and

oral operations, as well as cranial nerve injections, without relief, as might be expected, since the true pathology lies at a distance or below the point of actual pain reference.

This may be one of the reasons for the unsatisfactory results obtained in attempting to relieve such pain by treatment of the sinuses, the septum and teeth as well as by the use of injections of the peripheral branches of the nerves. Little effect upon such distant pathology can be expected.

A careful and complete study of the patient, suffering from atypical neuralgia is required to determine the location of the pathology. This should include roentgen studies of the teeth, mastoid, sinuses and chest, along with a careful history regarding former upper respiratory tract infections and a detailed thoracic, abdominal and pelvic examination. Too frequently the problem is turned over to the otolaryngologist whose attention, examination and zone of attack are confined to the area of referred pain about the face and head, without regard to the probable underlying visceral pathology.

A clear concept as to the origin of this pain will lead us to a more rational means of treatment. The careful search for signs of old sinus or mastoid disease is important. The possibility of referred pain from lower levels, caused by thoracic and visceral diseases must not be overlooked.

SUMMARY

Chronic attacks of deep, dull, aching pain in the eye, malar region, about the ear and down the neck, worse at night, throbbing in character, have been found associated with tenderness to deep pressure over the arterial branches of the neck and head.

This "carotidynia" can be elicited best along the course of the carotid artery, especially at the bifurcation on the side of involvement. In some cases the subclavian and brachial arteries show a similar tenderness.

In many cases it has been possible to demonstrate during the acute stage of pan-sinusitis that pressure over this structure produces pain referred to the eye and into the region of the zygoma, similar to that complained of by the patient. This has served, in

doubtful cases, as a means of differential diagnosis between trigeminal and atypical neuralgia.

When an anisocoria to carotid stimulation is present and persists on the side of the atypical neuralgia, an organic fixed basis for this symptom complex is indicated above the carotid bifurcation, and serves to differentiate it from the functional and distant reflex vasomotor disturbances associated with a similar pain distribution.

It seems reasonably certain that we are dealing with an underlying vascular pain mechanism in atypical facial neuralgia, which is distinct from the trigeminal and cervical neuralgias. This type of pain probably finds its port of entry to the brain and consciousness through branches given off by the vagus, which associate themselves with the large cranial vessels, and travel with the sympathetic branches to their fields of distribution. Fibers from the lower cervical and upper thoracic cord, by way of the carotid sheath, are important pathways for this type of pain and become intermingled with the vagus group.

It seems probable that this sensory mechanism is distinct from the sympathetic system, both as to origin and function, though the interrelations of pain and sympathetic responses are not unlike what is noted in other areas of the body. The direct or secondary vasomotor responses, associated with congestion and hyperemia, are also characterized by this type of pain, and the symptom complex of atypical neuralgia appears to be a syndrome of vascular pain.

Certain thoracic and visceral disturbances may find a peripheral reference of pain reflexly, through the vagus arc, as this structure supplies sensory fibers by way of the arterial branches to the deep structures of the face, neck, thorax and abdomen.

Following the various combinations of sensory, sympathetic, vagal and vascular sheath resections, an analysis of the results (assisted by a process of negative elimination) indicates the existence of three distinct pain mechanisms to the structures of the head, face and neck.

1. Superficial pain by way of the trigeminal and cervical branches.

2. Deep pain sense transmitted through the sensory branches and connections of the vagus nerve.

3. Tenderness and deep pain along the arterial tree through the carotid sheath to the cervical thoracic cord.

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BIBLIOGRAPHY.

1. Brouwer, B.: Pathology of Sensibility. Calif. and West. Med. J., *25*:329-330, Sept., 1926.

2. Brouwer, B.: Centripetal Systems in Brain as Influenced by Centrifugal Stimuli. Psychiat. en Neurol. Bl., *32*:320-327, May and August, 1928.

3. Cobb, S.: Cerebral Circulation, Quantitative Study of Capillaries in Hippocampus. Arch. Surg., 18:1200-1209, April, 1929.

4. Davis, Loyal E.: The Deep Sensibility of the Face. Arch. Neurol. & Psychiat., 9:283-305, March, 1923.

5. Fay, Temple: Atypical Neuralgia. Phila. Neurol. Soc., Jan. 28, 1927. Arch. Neurol. and Psychiat., 18:309-315, August, 1927.

6. Fay, Temple: Observations and Results from Intracranial Section of the Glossopharyngeus and Vagus Nerves in Man. Jour. Neurol. and Psychopath., 8:110-123, Oct., 1927.

7. Foërster, O.; Altenburger, H., and Kroll, F.W.: Über die Beziehungen des Vegetativen Nervensystems zur Sensibilitat. Zeitschr. f. d. Ges. Neurol. u. Psychiat., 121-139-185, 1929.

8. Forbes, H. S., and Wolff, H. G.: Cerebral Circulation: Vasomotor Control of Cerebral Vessels. Arch. Neurol. & Psychiat., 19:1057-1086, June, 1928.

9. Halphen, Monbrun, and Tournay: The Cephaleas in Oto-Neuro-Ophthalmology. Rev. D'Oto-Neuro-Ophthaimologie, 7:161-244, March, 1929.

10. Hassin, G. B.: The Nerve Supply of the Cerebral Vessels; Histologic Study. Arch. Neurol. & Psychiat., 22:375-391, Aug., 1929.

11. Herrick, C. J.: Introduction to Neurology. W. B. Saunders Co., pp. 249-262, 1916.

12. Hunt, Ramsay: The Sensory System of the Facial Nerve and Its Symptomatology. J. Nerv. & Mental Dis., June, 1909.

13. Hunt, Ramsay: On Herpetic Inflammation of the Geniculate Ganglion. A New Syndrome and Its Complications. J. Nerv. & Mental Dis., February, 1907.

14. Hunt, Ramsay: Otalgia Considered as an Affection of the Sensory System of the Seventh Cranial Nerve. Arch. Otol., 360; June, 1907.

15. Lapinsky, M.: Viszeral Bedingbe Genicksteifigkeit und Gleichgewichtsstorung. Med. Welt, 1:1306, October, 1927.

16. Lapinsky, M.: Arch. f. Psychiat., 80:599-623, 1927.

17. Ranson, S. W.: Unmyelinated Nerve Fibers as Conductors of Protopathic Sensation. Brain, 38:381, 1915.

18. Ranson, S. W., and Billingsley, P. R.: Afferent Spinal Paths and the Vasomotor Reflexes. Amer. J. Physiol., 42:16, 1916.

19. Sears, W. H.: Otogenic Paralysis of the Abducens with Especial Mention of Isolated Palsy Associated with Irritation of the Gasserian Ganglion. Trans. Amer. Laryng., Rhinolg. and Otolog. Soc., 1925.

20. Sherrington, Charles S.: Cutaneous Sensations. Schaefer's Physiology, II, 1000, 1900.

21. Spiller, William G.: Jour. Nerv. & Mental Dis., p. 736, 1906.

22. Spiller, William G., and Fay, Temple: Sensory Studies on the Face and Neck. Presented before the American Neurological Society, June, 1926 (unpublished).

23. Wilkinson, George: J. Laryngol., Otol. and Rhinol., 29:409, 1914.

LXXXI.

ANATOMIC STUDY OF EXTENSION OF A MASTOID INFECTION INTO THE DIGASTRIC MUSCLE AND ADJACENT STRUCTURES.*

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The muscular attachments to the surfaces of the mastoid tip are of paramount importance to otologists. At times infection extends beyond the confines of the cells with invasion of adjacent structures. This extension is apt to occur in an inward direction, causing those well known complications which all otologists dread: perisinous abscess, sinus thrombosis, extradural abscess, labyrinthitis, meningitis, abscess of the temporosphenoidal lobe or of the cerebellum. (Fig. 1.)

Extension outward is far less common. The reason is apparent if the anatomy of the mastoid bone is recalled. First, the greater thickness of the external cortex causes the infection to travel inward, destroying the thinner internal bony plate. Second, a fair percentage of mastoids are sclerotic or diploetic, thereby increasing the resistance to external invasion. Third, the vascular drainage by way of the veins is inward, carrying infection to the great vascular structures of the cranial cavity.

Infection extending externally usually manifests itself by a subperiosteal abscess, zygomatic abscess or an invasion of the muscles attached to the tip of the digastric groove. These muscles are the sternomastoid, that great muscular and surgical landmark of the neck, and the digastric, a muscle of great importance in the surgical anatomy of the upper neck as well as in the anatomic and functional adjustment of the hyoid bone.

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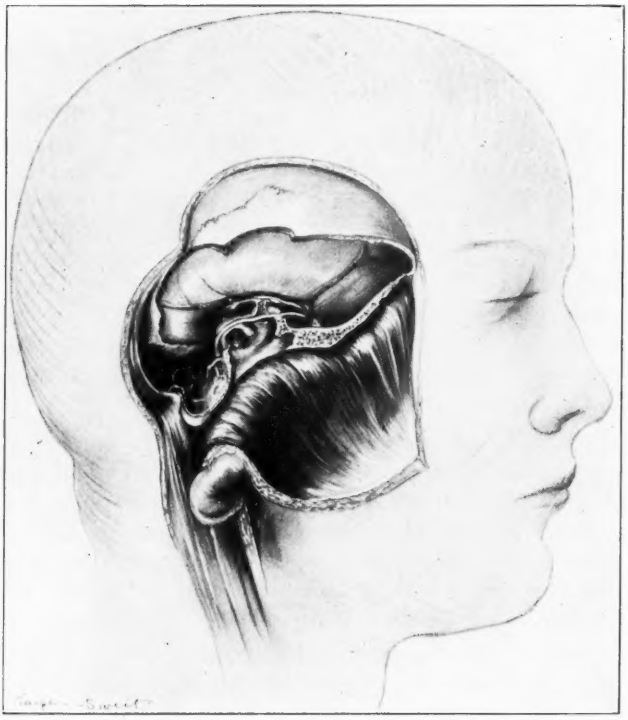

Fig. 1. Wet specimen dissection by the author showing mastoid dissection with anatomical demonstration of pneumatic tip cells, digastric ridge, sinus plate, facial nerve, geniculate ganglion, major petrosal nerve, gasserian ganglion, middle meningeal artery with anterior and posterior branches, meninges and paralabyrinthine cells. Also oval window, uncapped promontory and Trautmann's triangle.

Between the mastoid tip proper and a so-called inner tip is the digastric groove. (Fig.2.) This corresponds to an internal mastoid anatomic landmark which I have named the facial tip ridge, though it is commonly called the digastric ridge. It extends from the lower extremity of the facial canal backward and downward, separating the external from the internal tip cells. (Fig. 1.) This ridge is of surgical importance; for if a mastoid infection should extend to the external structures by way of the cells internal to it, the digastric muscle might be involved; though usually such invasion affects the sinus, the bulb or the facial canal. (Fig. 1.) If the extension is by way of the cells external to this ridge the digastric muscle is at times involved, but more often the sternomastoid with its facial sheath is invaded. (Fig. 1.) Anatomically, one might expect that the digastric muscle would be involved as often, if not more frequently, than the sternomastoid muscle, in extension from external tip cells. From both clinical and pathologic observations this is not the case. The reason for this is apparent when dissections of the mastoid, its tip and muscular attachments are made in a series of temporal bone dissections.

In an anatomic research of eight cadaver heads and six dry skulls, carefully dissected (a total of twenty-eight mastoids studied), approximately 60 per cent of the internal portions of the external tip cells—that is, those structures in relation to the digastric ridge and digastric muscle, were found sclerotic or diploetic. In the approximately 40 per cent remaining, these structures were diploe-pneumatic or very definitely pneumatic in type. Twenty-five per cent of the total demonstrated a definite pneumatic characteristic, the cells being separated from one another by a very thin, tissue paper structure of bone. This condition occurred in regard to its relation to the digastric groove and muscle as well. (Figs. 3 and 4.) Due allowance must be made for the fact that all these observations were made on adult specimens and solely in regard to the relation of the tip cells with the digastric muscle and digastric ridge. They are not intended to present data on anatomic characteristics of other portions of the mastoid process or of those cells in relation to the insertion of the sternomastoid muscle or of those internal to the facial tip ridge.

Fig. 2. Dry specimen dissection by the author showing mastoid tip, digastric groove, so-called secondary tip, styloid process and occipital groove.

Following is a tabulated report of anatomic observations of twenty-eight mastoid bones studied with regard to the type of cells present in relation to the digastric ridge and muscle:

Specimens	No.	Age	No. Mast'ds	Tip Cells		% Type of Cells
Wet	8	Adults	16	sclerotic	4	25
				diploetic	6	38
				diploe-pneuma	3	18.5
				pneumatic	3	18.5
Dry	6	Adults	12	sclerotic	2	17 plus
				diploetic	4	33 plus
				diploe-pneuma	2	17 plus
				pneumatic	4	33 plus
Total	14	Adults	28	sclerotic	6	21 plus
				diploetic	10	35 plus
				diploe-pneuma	5	17 plus
				pneumatic	7	25 plus

For anatomic convenience the digastric muscle is described as being composed of two bellies: a posterior and an anterior. They are separated by a tendinous portion which passes through a pulley-like structure (the separated fibers of insertion of the stylohyoid muscle) and is anchored to the hyoid bone. Anatomically, the muscle is one structure, though the muscular fibers are modified histologically because of functional necessity into this so-called central tendon. The posterior belly originates in the digastric groove and serves as a very important anatomic and surgical landmark to the structures of the upper portion of the neck as well as to the sphenomaxillary fossa. It is in close anatomic relation to the stylohyoid muscle and the parotid gland. Beneath it are the great vessels of the neck as well as the ninth, tenth, eleventh and twelfth cranial nerves. Along its lower border is the hypoglossal nerve, and it is in close relation to the hyoid bone and superior laryngeal nerve. Anterior to it are some of the facial vessels. (Figs. 5 and 6.)

If a careful dissection is made of the muscles attached to the mastoid tip and digastric groove, as well as the structures arising from the styloid process, a very definite as well as interesting anatomic condition is demonstrated. The sternomastoid, enclosed as it is by a splitting of the first layer of the deep cervical fascia, is well protected from invasion other than from the tip cells.

Fig. 3. Dry specimen dissection by the author showing mastoid dissection, pneumatic tip cell, digastric ridge, facial ridge, sinus plate, external and posterior semicircular canals, uncapped, styloid process, long handle of malleus.

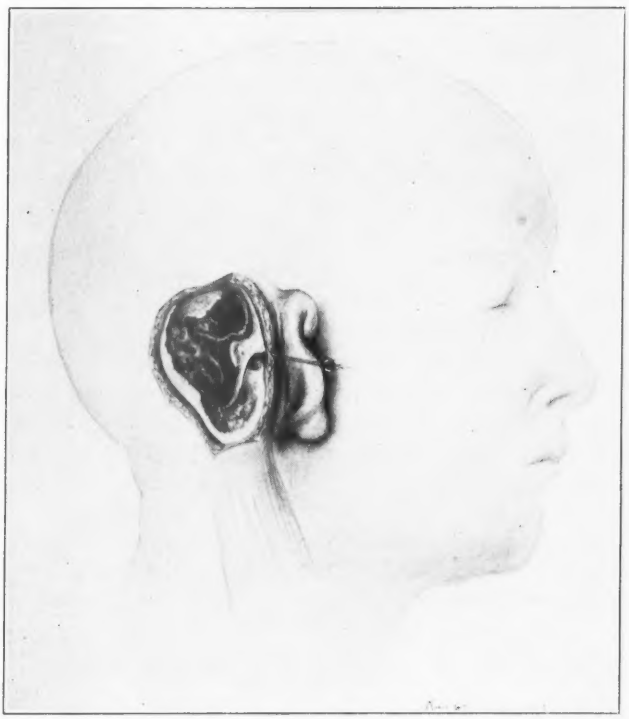

Fig. 4. Wet specimen dissection by the author showing diploetic tip, digastric ridge, facial nerve, external semicircular canal. Sternomastoid muscle.

The fascia covering the deeper layer of this muscle is highly resistant, markedly fibrous in character. The parotid gland is also enclosed in a fibrous capsule throughout, with the exception of its upper inner aspect. This capsule is also the result of the splitting of the first layer of the deep cervical fascia. The deeper layer of the fascial split is attached to the styloid process and extends to the angle of the mandible, serving as a ligament which in a measure protects the sphenomaxillary fossa from invasion. This anatomic arrangement of the fascia permits the posterior belly of the digastric muscle to assume a rather isolated position between the upper part of the sternomastoid, the parotid gland and the stylomandibular ligament. Apparently nature has ignored the posterior belly of the digastric muscle in so far as a very definite fascial covering is concerned. (Fig. 6.)

When infection extends into the digastric muscle from the mastoid tip cells there is only one direction for it to extend—that is, in the direction of the muscle, downward and forward, to a point where further extension or burrowing is possible. (Fig. 7.) This may be upward anterior to the stylomandibular ligament with involvement of the deep structures of the face or beneath the ligament to the sphenomaxillary fossa. In time such invasion may manifest itself by clinical evidence in the lateral pharyngeal wall. The usual direction is downward and superficial to the great vessels of the neck, ultimately burrowing beneath the sternomastoid muscle. The surgical axiom that pus under pressure may follow any direction is applicable here as in any other part of the body.

The close relation of the digastric muscle with a high percentage of pneumatic tip cells, in mastoids that at some time or other are very definitely involved and do not come to surgical interference, brings up an interesting possibility of invasion of the muscle without perforation of the tip cells—that is, by continuity of tissue. (Fig. 7.) This occurs around various joints in the body with the development of a fibrositis in the muscles. Why not in this location? From observation I am inclined to believe it is a definite clinical and pathologic entity, and that it has a bearing upon some pathologic conditions of the anterior neck.

Fig. 5. Wet specimen dissection by the author showing posterior belly of digastric muscle retracted demonstrating styloid process, parotid gland, facial nerve, stylomandibular ligament, bulb cells, semicircular canals, paralabyrinthine cells, promontory and inner middle ear wall uncapped.

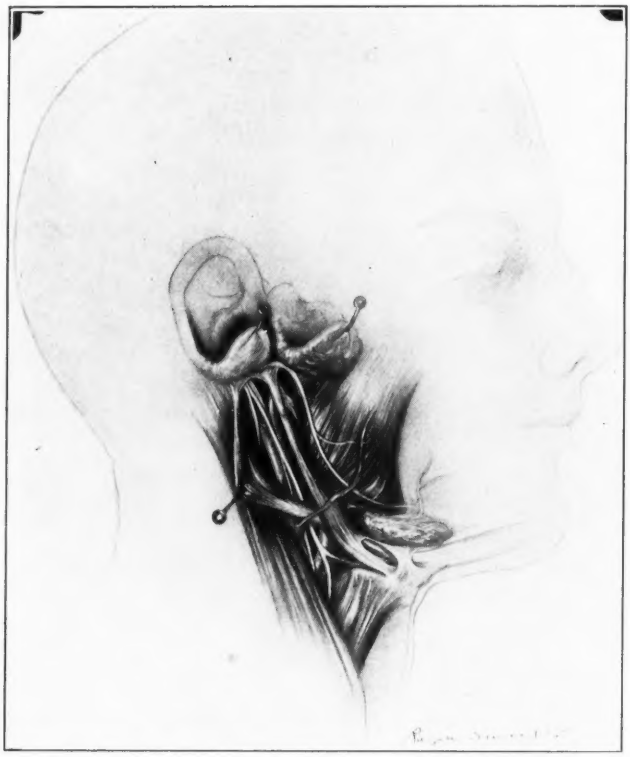

Fig. 6. Wet specimen dissection by the author showing sternomastoid muscle, everted parotid gland. Posterior belly of digastric muscle retracted showing its relation to the carotid sheath, stylohyoid muscle, stylomandibular ligament, facial vessels, tenth nerve, hyoid bone, submaxillary gland and superior laryngeal nerve.

Some of the vague anatomic misplacements of the structures of the anterior neck, especially the hyoid bone and larynx, may be explained on this hypothesis.

Certainly many of our mastoid infections that lie dormant in the tip cells, separated as they are by a tissue paper thickness of bone from this muscle, must have some pathologic as well as clinical effect upon the function of this muscle and parts under its influence. (Fig. 7.)

Most textbooks describe a Bezold abscess briefly. Many describe it as an extension of a mastoid infection into the sternomastoid.[1] In Bezold's original description[2] the paths of extension, both from an experimental and a clinical view, are definitely given. He does not confine this invasion specifically to the sternomastoid muscle. One case is described as extending into the sheath of the digastric muscle, though from anatomic observations I have found the sheath to be more imaginary than real, and not definitely discernible to the naked eye. The muscle itself lies in a sheathlike channel, formed by the cervical fascia and sheath coverings of contiguous muscles. I was unable to demonstrate a definite sheathlike covering to the muscle grossly without the aid of my imagination.

The French school recognizes a purulent infection as extending into the digastric muscle and call it "The Abscess of Mouret."[19] They insist upon its differentiation from a true Bezold abscess and say that its development is by way of the jugo-digastric cells. Their observations have been definitely corroborated by my anatomic studies.

Extension into the digastric muscle is characterized by marked preauricular swelling and pain. The submaxillary region is greatly swollen. This swelling may, and usually does, extend downward along the anterior border of the sternomastoid muscle. Great difficulty is encountered in opening the mouth and great pain is felt at the attempt. The patient had a rapid pulse, runs a high fever, and has a very definitely septic appearance. The condition must be differentiated from parotitis, parotid abscess, furunculosis and the usually described Bezold abscess. (Figs. 6

Fig. 7. Wet specimen dissection by the author showing external tip cells knocked off and relation of digastric muscle to digastric ridge, also direction of digastric muscle with sternomastoid muscle everted showing its inner fascial covering containing nerves and vessels.

Fig. 8. Wet specimen dissection by the author of temporomandibular joint, showing glenoid fossa, condyle, vaginal portion of styloid, styloid process, anterior and inferior portion of external bony auditory canal, petrotympanic or glasserian fissure. Mastoid process and digastric groove.

and 8.) When it occurs as a complication following a simple mastoidectomy it taxes diagnostic skill and surgical judgment. Fortunately in two suspicious cases that I attended procrastination served well; probably due to a feeling that a complete exenteration of the tip cells had been made. Even though a careful history and cautious examination of the ear and mastoid process is made, its differentiation from parotitis, parotid abscess furunculosis and the extension into other muscular structures, as described by Bezold in his original work, is frequently difficult.[2] One of the patients, whose condition I diagnosed as infection extending into the digastric muscle, demonstrated a much greater preauricular swelling with greater difficulty in opening his mouth than might be expected in other types of Bezold's abscess. Some swelling on the floor of the mouth and lateral pharyngeal wall was determined by palpation and inspection.

Treatment is strictly surgical by way of the anatomic approach known as the submaxillary, with drainage. Briefly, the technic is similar to that described by Mosher at the meeting of the American Academy of Ophthalmology and Otolaryngology in Atlantic City, and published in full in the proceedings of the Academy.[17]

Along the inferior border of the maxillary bone a T-shaped incision is made, the long arm of the T being directed downward along the anterior border of the sternomastoid muscle, exposing the digastric muscle, cautiously avoiding the hypoglossal nerve; locating the pus by sharp dissection if possible, otherwise using Hilton's method. Rubber tube drainage is used.

When attending my first patient suffering with this condition I was not acquainted with the method of approach advocated by Mosher for deep pus in the neck. The general surgical principle of seeking pus by following along the route from the point of origin was used.[2] It proved highly satisfactory, and an excellent result was obtained. Having since studied the anatomy and principles involved in the submaxillary technic I am convinced that this method would be far preferable and would involve less hazard to the facial nerve as well as decreasing the chance of invasion of healthy walled off tissue.

CONCLUSIONS.

1. Extension of a mastoid infection from the tip cells into the digastric muscle and adjacent structures does occur.

2. It is relatively uncommon, probably because of the excellent type of mastoid surgery that is usually performed at the present time.

3. It must be differentiated from furunculosis, parotitis and parotid abscess.

4. Simple inflammatory extension into the digastric muscle from mastoid infection probably does occur, causing a functional disturbance of this muscle and allied structures.

5. The treatment is surgical by way of the submaxillary approach.

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BIBLIOGRAPHY.

1. Beck, Joseph C.: Applied Pathology in Diseases of the Nose, Throat and Ear. St. Louis, C. V. Mosby, 1923, pp. 82-83.

2. Bezold, Fr.: Ein neuer Weg für Ausbreitung eitriger Entzündung aus den Räumen des Mittelohrs. Deutsche med. Wchnschr., 1881, VII, 381.

3. Bezold, Fr.: Leiner statistische Zusammenstellungen. Zeitschr. f. Ohrenheilk., 1903, XLIII, 273.

4. Bezold, Fr., and Siebenmann, Fr.: Textbook of Otology. Chicago, E. H. Colegrove, 1908, pp. 183-184.

5. Botella, E.: Consideraciones sobre 13 casos de mastoiditis de Bezold. Rev. clin. de Madrid, 1910, III, 361-369.

6. Castex, A.: Mastoidite de Bezold et méningite. Bull. de laryngol., otol. et rhinol., 1908, XI, 109.

7. Clay, J. V. F.: Case of Bezold's Mastoiditis. J. Ophth., Otol. and Laryngol., 1927, XXXI, 291-293.

8. Eisinger, K.: Bezoldmastoiditis mit Fazialislähmung in allen drei Aesten; Operation; Rückgang des Fazialis; Heilung. Monatschr. f. Ohrenheilk., 1925, LIX, 863.

9. Fernandez, R.: Un cas de mastoidite de Bezold avec extension sous occipitale. J. de chir. et ann. Soc. belge de chir., 1910, X, 401-404.

10. Keraudren: Cholesteatome volumineux de la mastoïde; mastoïdite de Bezold; paralysie faciale; evidement pétro-mastoïdien. Clinique, 1913, VIII, 87.

11. Kerrison, Philip D.: Diseases of the Ear. Phila., Lippincott, 1921, p. 187.

12. Kopetzky, Samuel J.: Otologic Surgery. New York, Hoeber, 1925, pp. 22, 121.

13. Laudry, M., and Billard, J.: Bezold's Mastoiditis with Perforation Into Pharynx: Case. Acta oto-laryng., 1927, XI, 471-473.

14. Lake, Richard: Contributions to the Art and Science of Otology. London, Macmillan, 1926, p. 40.

15. Levine, Morris: Practical Otology. Philadelphia, Lea & Febiger, 1927, pp. 209, 252.

16. Milligan, W., and Wingrave, Wyatt: Practical Handbook of Diseases of the Ear. New York, W. Wood & Co., 1923, pp. 149-150.

17. Mosher, H. P.: The Submaxillary Fossa Approach to Deep Pus in the Neck. Trans. Am. Acad. Ophthal. and Otolaryng., 1929, XXXIV, 19-36.

18. Politzer, Adam: Textbook of the Diseases of the Ear, 6th ed., rev. by Milton J. Ballin. London, Baillière, Tindall & Cox, 1926, pp. 402, 516.

19. Portmann, Geo., Bordeaux, France: Personal communication.

20. Shaheen, H.: Bezold's Mastoiditis Associated with Mental Symptoms; Operation; Recovery. J. Laryngol. and Otol., 1928, XLIII, 580.

21. Tod, Hunter: Diseases of the Ear. Oxford Univ. Press, 1926, pp. 219, 236-237.

22. Whale, H. L.: Von Bezold's Mastoiditis. J. Laryngol., 1916, XXVI, 520.

23. Whiting, Frederick: Modern Mastoid Operation. Philadelphia, Blakiston, 1905, pp. 163, 169.

24. Ziegelman, E. F.: Classification of Author Based Upon Gross Anatomical Appearance of Mastoid Bone at Time of Dissection. loc. cit.

LXXXII.

RECURRENT HOARSENESS AND APHONIA WITHOUT DEMONSTRABLE LARYNGEAL PATHOLOGY.*

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In offering this brief discussion, we feel to apologize for not having made a more comprehensive survey of the literature to determine if credit should be given for its having been previously reported by other observers. However, the annoyance experienced in not being able to relieve these patients of what seemed to be such a simple thing made us feel that any possible solution of the problem involved justified its presentation, for discussion at least.

Perusal of a limited number of textbooks—some recent, some older—appears to indicate that recurrent attacks of hoarseness and aphonia, without demonstrable change in the larynx, are supposed to arise from various more or less indefinite causes. The only other point upon which the accounts agree is that hysteria qualifies as the preponderant etiology.

DeSchweinitz and Randall,[1] in their book published in 1901, offer the following, under a heading of laryngeal spasm in adults:

"While laryngeal spasm in children presents a fairly uniform clinical picture and is due apparently to a limited number of causes, the opposite holds true in adults. Notwithstanding its comparatively infrequent occurrence, we find cases reported as due to hysteria, to various local lesions of the nose and throat, to reflexes from other organs, and to lesions of the nerve trunks and centers. It may occur at any time of the night or day, or under various conditions of health or disease. It may be phona-

*Read before the Utah State Oto-Ophthalmological Society, Salt Lake City, March 21, 1932.

tory, deglutitory or respiratory. . . . Epilepsy, chorea, tetanus and hydrophobia are the general nervous affections of which laryngeal spasm is the more or less constant accompaniment. As Gottstein remarks, it occasionally occurs in cases in which no explanation can be found."

Further, under the headings of inco-ordination of the laryngeal muscles, and dysphonia spastica, the same authors state:

"This is a curious laryngeal neurosis which has occasionally, although rarely, been noted. It seems to be in most cases a manifestation of hysteria; and in some cases has occurred from overfatigue of the larynx in preachers. Any attempt at phonation results in a more or less complete closure of the glottis so that not a sound comes forth; and with it may be associated some respiratory spasm—that is, the adduction of the vocal cords lasts long enough to cause inspiratory dyspnea. In other cases, there comes a high-pitched, shrill note from the larynx, possibly articulated, but not long sustained. . . . With the laryngoscope, no abnormality is to be observed until the patient attempts to phonate, when the vocal cords are seen to be firmly apposed in median line."

These authors also report a case in which there was continuation of periodic attacks of hoarseness and aphonia lasting many years after a serious general illness, with negative laryngeal findings.

Sir St. Clair Thomson[2] makes the following observations:

"Phonic spasm, or dysphonia spastica, is a rare affection which has been compared with writer's cramp. It occurs in professional voice users of nervous temperament, who, on attempted phonation, are seized with a spasmodic contraction of the adductors and tensors of the vocal cords. The spasm may cease, when the attempt at phonation is abandoned. B. Fraenkel gave the name of mogiphonia to that form of spasm which only occurs on attempts at singing or public speaking, but does not interfere with ordinary conversation. . . . Treatment is apt to be disappointing."

Discussing a condition which he called "Follicular Pharyngitis," D. Braden Kyle mentions "A chronic inflammatory condi-

tion of the pharyngeal mucosa, especially involving glandular structure. It is characterized by an altered secretion and by irritation of the pharynx, accompanied frequently by a sharp, hacking cough. Alteration of the voice is a constant symptom, varying under different circumstances from a slight hoarseness to complete aphonia.

"The influence of occupation is a most important one, those who are compelled by their vocation to use the vocal apparatus under many and varied unfavorable conditions being especially liable to the acquirement of the malady. Thus, it is peculiarly a disease of clergymen, who, in addition to their Sunday services, are taxed by other public demands; of lawyers, with practices necessitating long and fatiguing pleas in dusty and ill ventilated courtrooms; of campaign speakers, actors, singers and of those in the host of minor callings, such as hucksters and auctioneers. The condition is but another phase of simple chronic pharyngitis. . . . . The voice, on the slightest use beyond a limit peculiar to each case, becomes hoarse and muffled, its quality is altered, and it may fall to a mere harsh whisper or even complete aphonia. . . . ."

In the third edition of French's "An Index of Differential Diagnosis of Main Symptoms," E. Farquhar Buzzard wrote: "In cases of hysteria, a functional aphonia is by no means uncommon, and in many cases can be distinguished from aphonia due to organic disease only by an examination of the larynx. Hysterical aphonia may be complete; in other cases the voice is reduced to a whisper, and yet the patient is able to adduct the cords properly in coughing. This is sometimes a recurrent malady, and the suddenness of its onset, as well as the suddenness with which it is often cured are characteristic."

From these several brief quotations, one may gather that there is a recurrent dysphonia, sometimes amounting to aphonia, the etiology of which is still in doubt. Hysteria is acknowledged by most authors as a factor of great likelihood in accounting for these attacks; and for those instances of sudden onset or sudden termination, perhaps must still be allowed. Our present interest centers around those of more gradual development, in which hys-

teria is not, we believe, a factor. Kyle considered them to be a result of chronic simple pharyngitis. All have recognized the recurrent nature of the malady, usually without apparent reason, unless overuse of the voice could be adduced as having incited the reaction. Hysteria, like "catarrh," has achieved decided threadbareness as a disguise for our diagnostic incompetency; and sincere study or analysis of any practitioner's experience can only lead to a belief that there is almost invariably an organic basis for any condition one is tempted to so label. Otolaryngology has already contributed its share of specimens to the figurative osteologic closet exhibits, of which globus hystericus is an outstanding example. We believe "hysterical dysphonia and hysterical aphonia" belong in the same category.

There seems no doubt that these attacks are reflex, however, and perhaps spastic in nature; but we have not been able to discover any visible deviation from the normal in endolaryngeal appearance or in readiness, manner or degree of approximation of the vocal cords during attempts at phonation. Some writers[5] attribute the mechanical production of disturbance in phonation to gross failure in approximation of the cords, stating that when these patients attempt to phonate the bands are in firm contact in their anterior three-fourths, with a triangular separation posteriorly. This observation, as well as the one that the cords are approximated in coughing but remain immobile during attempts at phonation, have not been verified in our patients.

That the individual can approximate the vocal bands for coughing would seem to have little significance relating to vocalization, since narrowing of the rima glottidis by action of the transverse and oblique arytenoid muscles alone, or even closure of the so-called sphincter of the laryngeal vestibule, would certainly produce sufficient resistance to the massive air column for coughing. The production of voice, on the contrary, is normally the result of concerted and finely co-ordinated action of all the adductors at least. The very necessity for this precise co-ordination of several factors establishes a vulnerable point of attack for reflex disturbance. It is not uncommon to see persons who, regardless of whether they speak little or much, have days when, as they become fatigued from nervous strain and tension-producing work,

tend to vocalize in the falsetto range or to become actually hoarse. Yet most of these individuals would, because of this alone, scarcely be classed by anyone as likely material for manifestations of hysteria.

All of us, no doubt, have seen patients thrown into sudden laryngeal spasm and stridor, from application to the vault of the nasopharynx of some sharply pungent or astringent medicament, even where there was no possibility of any of it having contacted laryngeal tissues. The etiologic culpability of large or diseased adenoids in causing spasmodic croup among children is no longer to be questioned. Hoarseness as one of the early symptoms of neoplasm in the nasopharynx is detailed by French K. Hansel.[6]

Such observations remove all doubt as to the presence of some intimate association or reflex possibility between the larynx and the postnasal region. A study of anatomic relationships existing between distributions of branches from the superior cervical ganglion of the sympathetic, the vagus nerve,[7] and from the sphenopalatine ganglion further attests the likelihood of reflex association between nasal tissues and the larynx. Certainly, impulses that lead to sneezing have their usual origin in the nose, and the occurrence of cough from nasal irritation is probably more common than is ordinarily recognized. The fact that certain asthmatic seizures have their nidus of origin in the nose, and are controllable, through the sphenopalatine ganglion, has long been understood,[8 9] and it would appear at least possible for other reflex conditions involving the lower respiratory tract to have their beginning there.

Study of our patients presenting the phenomenon of recurrent hoarseness without demonstrable pathology in the larynx, at first led us to believe the condition must result from postnasal drainage actually entering the larynx and setting up an irritation there. This position we were forced to abandon, after repeatedly seeing patients spontaneously recover normal voices, with not a whit of variation observable in the amount or character of discharge, and after frequently observing individuals with profuse postnasal drainage from maxillary sinuses—some of which could at times actually be seen clinging to intralaryngeal surfaces—who had no

history of any vocal disturbance. Experience with the following patient first directed our attention to ethmoid disease as a possible etiology of these troubles.

Mrs. G. W., aged 41, school teacher, came complaining of frontal and parietal headache, together with marked dysphonia. Headache had annoyed her much of the time for several years. The hoarseness occasionally amounting to complete aphonia, had recurred many times and for varying periods, but had never been associated with any particular sore throat. There was some complaint of postnasal drainage, although the amount of discharge did not seem great. Cough was not troublesome in degree at any time. Examination of this patient revealed large middle turbinates crowding ethmoids and septum on both sides and each bathed in mucopus. Mirror laryngoscopy discovered no intralaryngeal pathology, and movements of the vocal cords seemed entirely normal. Otherwise, the examination was negative.

Believing her headache due to nasal pathology, we thinned out the middle turbinates by the simple expedient of crushing them, which—since both were largely pneumatized—gave complete freedom from pressure. As each side was done, we noted a considerable drainage of mucopus from the ethmoid cells, and decided to refrain from further surgical interference at that time. Within a few days the headaches ceased, and have not returned in a period of more than two years. Much to our surprise, the dysphonia also rapidly cleared up, and she has had no further attacks of even mild hoarseness.

At the time of obtaining this unexpected result, we had under our care a district judge, who several times each year was incapacitated for his work by attacks of aphonia. Each of these would begin with a mild hoarseness, increasing over a week or ten days to complete loss of voice. After a variable period, recovery would gradually take place. We had removed the patient's tonsils because of repeated tonsillitis, and also in the hope that they had something to do with causing the voice disturbance. Examination of the larynx was always negative, so far as revealing any visible pathology there. Considerable cough was at times associated with the dysphonia, bothering particularly when he laid down to sleep

and upon first arising in the morning. Study of the nasal condition demonstrated negative maxillary, frontal and sphenoidal sinuses, but showed some evidence of hyperplastic ethmoiditis. Operation of the ethmoids, after the method of Pratt, was done and marked disease found on both sides. The operation was performed in January, 1931, since which time the patient has had no further hoarseness or cough.

Gratifying results achieved in these and other similar instances have led us to study carefully for intranasal disease—especially of the ethmoids—every patient complaining of recurrent hoarseness or aphonia, and where intralaryngeal pathology is not found to account for the trouble. While our study does not as yet include any large series of these cases, we have yet to see the first one rightly falling into this class which has not been completely relieved of the recurrent voice disturbance by having diseased ethmoids cleaned out and cared for. In several patients who were averse to nasal surgery we have tried various other means without having any appreciable influence upon frequency or duration of the attacks.

We are, of course, fully cognizant that, with this as with all things in medicine, there will be exceptions, and that certain definite limitations must apply. However, we believe the results already obtained have been sufficiently favorable to warrant offering the hypothesis that chronic ethmoiditis is often the underlying etiology of recurring hoarseness and aphonia without demonstrable pathology in the larynx, and which has usually been considered as of hysterical or other cryptogenic origin.

BIBLIOGRAPHY.

1. DeSchweinitz, G. E., and Randall, B. Alex.: An American Textbook of Diseases of the Eye, Ear, Nose and Throat. Saunders, 1901, pp. 1156-7-8.

2. Thomson, Sir St. Clair: Diseases of the Nose and Throat. Appleton, 1928, p. 609.

3. Kyle, D. Braden: A Textbook of Diseases of the Nose and Throat. Saunders, 1916, pp. 566-7-9.

4. French, Herbert: An Index of Differential Diagnosis of Main Symptoms. Wm. Wood & Company, 1917, p. 628.

5. Jackson, Chevalier, and Coates, George Morrison: The Nose, Throat and Ear, and Their Diseases. Saunders, 1929, p. 772.

6. Hansel, French K.: Malignant Tumors of the Nasopharynx with Involvement of the Nervous System. Archives of Otolaryngology, October, 1931, p. 530.

7. Gray's Anatomy. Spitzka, p. 1068.

8. Sluder, Greenfield: Headaches and Eye Disorders of Nasal Origin. Mosby, 1918, p. 210.

9. Byrd, Hiram: The Law of Transmission of Nasal Disturbances. Eye, Ear, Nose and Throat Monthly, April, 1924, pp. 139-140.

10. Pratt, Fred J., and Pratt, John A.: Intranasal Surgery. F. A. Davis Co., 1925, p. 214.

35 EAST 2ND SOUTH.

LXXXIII.

OBSERVATIONS ON THE TEMPORAL BONE.*

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Montreal.

Anatomic investigation of the internal ear and its relationship to neighboring structures is at best carried out with difficulty. The subject can be approached by three methods, none of which proves entirely satisfactory, namely, gross dissections, microscopic sections and corrosion casts. Specimens obtained by dissection lose their relative position; microscopic sections, although revealing the finer details, lose the general outline of the whole organ, while corrosion preparations show only the bony cavities, the membranous network being destroyed.

The anatomy of the labyrinth in mammals has been carefully studied by Denker and Alexander,[1] by means of corrosion casts, while in more recent years, in the same manner, Siebenmann and Korner obtained preparations of the human temporal bone.

As decribed in Siebenmann's work, the specimens are injected with fusible metal: (a) "immediately on removal of the bone from the body, without previous treatment, and (b) after being treated with some fixing agent, such as formalin, and then dried previous to injection."[2]

Korner's method consists of macerating the bone, removing the soft parts and drying thoroughly before preparing the bone for the lead. When the soft tissues are left in place the injection of the metal is made through an artificial opening in the mastoid cortex. According to Siebenmann's method, the ramifications of the mastoid cells and their continuity with the middle ear and eustachian tube are beautifully shown. The directions in which

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From the Department of Otolaryngology, Royal Victoria Hospital, and the Department of Anatomy, McGill University.

The corrosion casts photographed are in the McGill Anatomical Museum.

All photographs show actual size of specimens except Nos. 1 and 5. These are slightly enlarged for the sake of clearness.

infection is likely to spread are clearly seen, but the labyrinth itself does not fill with metal. This is due to the fact that the footplate of the stapes with its attached ligament fits snugly into the oval window and prevents the molten lead from entering.

The macerated bone (Korner) gives the most complete casts, as the minute cavities throughout the temporal bone, including the labyrinth, become filled with the metal.

The metallic casts about to be described were made with the object of demonstrating greater detail and showing clearly certain well known anatomic features.

Injection of fusible lead (Wood's metal) and the subsequent method of dealing with the macerated bone have been fully described elsewhere.[3] [4]

Particular attention was given to the following anatomic details: The course of the facial nerve, the acoustic nerve with its ampullary branches, the accessory ampulla of the horizontal semicircular canal, the perilymphatic duct, and the endolymphatic duct with its sac.

The Facial Nerve.—In corrosion casts, as a rule, the facial canal is so embedded in the substance of the mould that it is lost to view, after passing through the internal acoustic meatus. With this in mind, an attempt was made to free the canal in its entire length from the bottom of the internal acoustic meatus laterally to the geniculate ganglion, along the horizontal portion first backward and outward and then downward to the stylomastoid foramen.

This was done in thoroughly macerated and dried temporal bones by means of thin wires. The internal acoustic meatus was carefully cleaned and all the foramina opened. The upper anterior depression contains the internal opening of the facial or fallopian canal and easily admits heavy piano wire. In the posterior funnel-shaped depression there are several delicate openings through which the terminal vestibular branches of the acoustic nerve run to the ampullæ of the superior and horizontal semicircular canals.[5]

The lower depression anteriorly shows a variable number of minute openings which differ from the foramen above in that

they admit only fine piano wire. These foramina carry the cochlear nerve directly to the basal turn of the cochlea. Somewhat behind is situated the foramen singulare, which transmits a lower terminal branch of the vestibular nerve to the ampulla of the posterior semicircular canal. No mention is made of the acoustic branches to the utricle and saccule, as they do not appear in metallic casts.

Figure 1 is a superior view showing a metal cast of an adult left temporal bone. The course of the facial and acoustic nerves (seventh and eighth) can easily be traced through the internal acoustic meatus on the interior of the skull outward. The acoustic nerve will be considered first as it is of lesser importance from

Fig. 1.

a surgical point of view. The vestibular branch of the acoustic nerve runs posteriorly and the upper terminal branches can be seen together, passing to the ampullæ of the superior and the horizontal semicircular canals. The lower terminal branch through the foramen singulare is seen going to the ampulla of the posterior canal. Below the facial is the cochlear branch of the acoustic nerve and it runs anteriorly into the base of the cochlea.

The facial nerve, as is well known, consisting of a motor and sensory root (pars intermedia), passes horizontally outwards in the anterior superior portion of the internal acoustic meatus for about 10 mm. and then turns slightly forward to the geniculate

ganglion. This is as far as we can follow it in this direction, as the branches from the ganglion, especially the greater and lesser superficial petrosal nerves, leave the temporal bone at this point. At the geniculate ganglion, or so-called knee, the nerve turns sharply backward and can be seen to disappear under the horizontal semicircular canal.

Figure 2 shows a lateral view of the same cast, and the remaining portion of the facial nerve in the temporal bone can be followed without difficulty. From the geniculate ganglion, just above the center of the cochlea, the course of the nerve lies horizontally and it "continues backwards along the inner tympanic wall above the fenestra ovalis."[6] Just above and behind the oval window

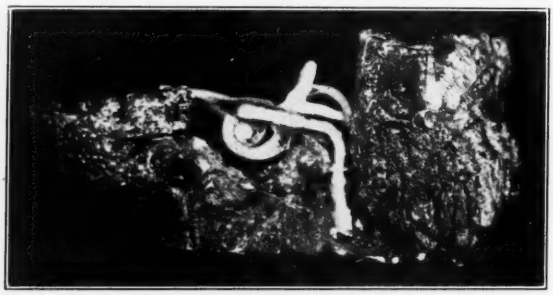

Fig. 2.

the nerve runs parallel to and just below the horizontal semicircular canal. When this transverse portion, measuring 10 to 12 mm., reaches the border between the posterior and inner walls of the tympanic cavity, and identified on casts by the vertex of the horizontal canal, it takes a sudden turn downward. The vertical part of the facial nerve runs downward for another 10 to 12 mm., to end at the stylomastoid foramen.

Although we speak of the "horizontal" and "vertical" portions of the facial canal, they are not exactly horizontal and vertical in direction.[7]

By drawing an imaginary line obliquely through the center of the spine of Henle, so that it forms an acute angle anteriorly with the sharp upper edge of the zygoma of about 15 degrees, we have

Fig. 3.

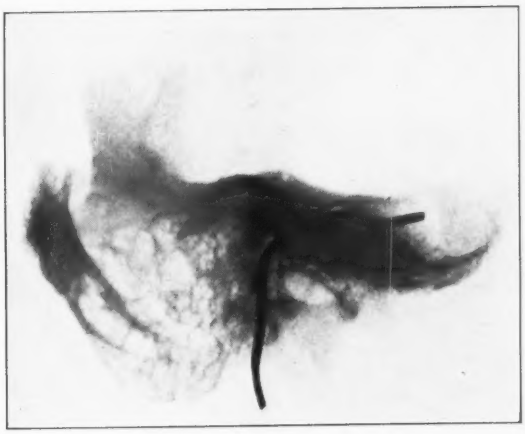

Fig. 4.

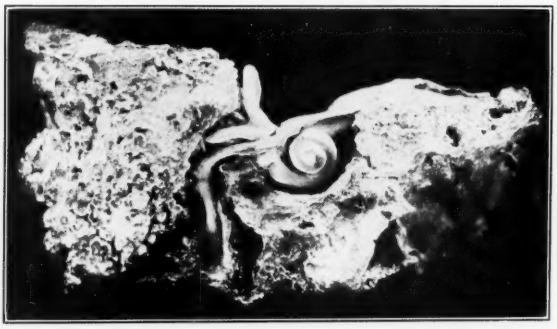

Fig. 5.

a fairly accurate indication of the direction of the horizontal portion of the nerve upon the inner wall of the tympanum.

Again, for the descending portion, if a line passes vertically through the center of the spine of Henle above, and is in contact with the anterior border of the mastoid process below, it can be taken as a reliable guide to the course of the vertical part of the facial nerve, from the downward turn just beneath the floor of the aditus to its emergence through the stylomastoid foramen.

The Accessory Ampulla.—An interesting occurrence in the labyrinth, more from an anatomic than a physiologic point of view, is the presence of an accessory ampulla. This is a rounded

Fig. 6.

swelling at the posterior end of the horizontal semicircular canal and first described by Albert A. Gray. It is easily demonstrated on corrosion casts, although present in only slightly more than half the specimens examined.

The accessory ampulla has the same symmetrical appearance as the more constant anterior one adjacent to that on the superior semicircular canal. The function of this ampulla is obscure, as it seems to be devoid of nerve structure on microscopic section.

Whether it is a vestigial remainder, as suggested by Gray, of "a fusion of the posterior end of the horizontal canal with the ampullary end of the posterior canal,"[8] a condition found in certain mammals, as the leopard, horse and polar bear, further investigation alone will disclose.

Fig. 7.

The Perilymphatic Duct.—This consists of a narrow, fairly straight canal of about 10 mm. in length, which simulates an inverted funnel in appearance. Beginning above in the floor of the scala tympani, about 1 or 2 mm. from the round window, the bony canal extends downward, forward and inward, and emerges in an enlarged opening on the inferior surface of the temporal bone close to its posterior edge. The triangular opening of the duct is bounded posteriorly by the jugular bulb, laterally by the internal carotid artery and medially by the posteroinferior border of the petrous portion of the temporal bone.

The perilymphatic duct, as the name implies, is in communication above with the perilymph of the scala tympani, while below it continues inward through the jugular foramen and dura mater, to open directly into the subarachnoid space within the skull. The importance of this narrow channel lies in the fact that under certain conditions it is a dangerous pathway of infection between the labyrinth and the cerebrospinal fluid of the cranial cavity. The size, length and relationship to the surrounding structures are well seen in Fig. 7.

The Endolymphatic Duct.—This is a very fine, slightly curved tube about 5 mm. in length and extending from the vestibular part of the labyrinth to the posterior surface of the petrous portion of the temporal bone.

Fig. 8.

It commences as two extremely thin membranous ducts, one from the posterior wall of the saccule and the other from the anterior medial wall of the utricle. These unite to form a larger tube, although still very narrow in caliber, and leaving the labyrinth on its medial and upper surface it arches inward and backward, through the aqueduct of the vestibule, to the inner surface of the skull. Here it dilates, through a slitlike opening, into a blind sac which is situated about 7 mm. posterior to the opening of the internal acoustic meatus.

This closed dilatation varying from 10 to 15 mm. in length by 8 to 9 mm. in width, is flattened and lies in the substance of the dura mater extending backward towards the lateral sinus.

The direction and course of this fine tube is shown in Fig. 8, beginning at its opening in the vestibule, just above the vestibu-

lar nerve, to the ampulla of the posterior canal and close to the base of the common crus of the superior and posterior canals. The terminal expanded sac is also well demonstrated.

The endolymphatic spaces, comprised of the membranous canals with their ampullæ, the utricle and saccule with their connections, the membranous duct of the cochlea (scala media) and the endolymphatic duct with its sac, constitute an isolated system. It is, as stated by Professor E. Ruttin of Vienna, "entirely closed, having no communication with the perilymphatic spaces and no communication outside. Therefore, it does not form a direct route for meningitis unless the dura is ruptured, which is in contrast to the perilymphatic spaces."[9]

In conclusion, it might be mentioned that the above specimens received no further treatment after dissolving away the bone in hydrochloric acid, apart from painting the various structures. Close attention is necessary in preparations of this kind, especially when the injected bones are immersed in acid. Daily removal of the softened bone is essential; otherwise, if left in acid too long the finer canals are ruined. As stated previously, fine wire was passed into the nerve canals and ducts as far as possible, in order to assure their patency for the fusible metal. Even with the greatest of care and constant watching it is only on rare occasions that the perilymphatic duct, and particularly the endolymphatic duct with its terminal sac, appear in their whole length on corrosion casts.

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REFERENCES.

1. Alexander, G.: Arb. a. d. Anat. Inst., 19:571, 1902.
2. Siebenmann, F.: Korrosionsanatomie. Jena. Gustav Fischer, 1897.
3. Rogers, Lee: Annals of Otology, Rhinology and Laryngology, March, 1921.
4. Tremble, G. E.: Arch. Oto., Nov., 1926, Vol. 4, pp. 412-420.
5. Spalteholz, W.: Hand-Atlas of Human Anatomy, Vol. III, p. 727, 1923.
6. Politzer: Diseases of the Ear (Ballin). Sixth Ed., p. 25.
7. Kerrison, P. D.: Diseases of the Ear, p. 408, 1913.
8. Gray, A. A.: Jour. of Anat. and Phy., Vol. XXXIX, p. 349, 1905.
9. Personal communication to the author.

LXXXIV.

CLINICAL VERSUS X-RAY DIAGNOSIS OF SINUSITIS.*

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There is a possibility that the title of this presentation may be misleading, as I am using the "clinical versus X-ray diagnosis" only in my case reports, and I believe Dr. Widmann is following the same line of thought in presenting the skiagraphic slides. In reality, I wish to bring out as the essential factors: the rhinologist should be capable of making a correct interpretation of the roentgenogram; that his opinion may be unbiased, he should draw his own conclusions from the film before reading the roentgenologist's report; and the limitations of the X-ray film as a single factor in making a diagnosis.

Dr. Law,[1] in his excellent paper "Interpreting Sinus Roentgenograms," read before the Eastern Section here in Atlantic City last year, defined a roentgenogram as "A maze of delicate lines that have a definite meaning if rightly translated." He goes further and states, "Translation means study and comparison with the clinical evidence." This, to my mind, is the crux of the entire situation. No one should ever make a diagnosis from the X-ray plate without full clinical knowledge of the case, which can only be obtained by careful intranasal examination.

The rhinologist, after studying and evaluating all of the data —history, clinical and laboratory—should then study the roentgenograms and draw his own unprejudiced conclusions from them, and thereafter should read the roentgenologist's report, using that report as a check upon his own diagnosis. It is my conviction that the rhinologist should become and can become an expert in reading roentgenograms within his restricted field, and should use the information so obtained as a necessary and inval-

*Read before the thirty-eighth annual meeting of the American Laryngological, Rhinological and Otological Society in Atlantic City, May, 1932.

uable part of his diagnostic routine. The personal equation enters into the reading of the fine lines and shadows of the roentgenogram, and if the rhinologist's interpretation of the film does not agree with the roentgenologist's report, or if the clinical findings do not agree with the interpretations of the film, then a consultation must be held if the patient is to have his due.

When I speak of the rhinologist becoming an expert in the reading of the roentgenograms, I do not mean that he should be a roentgenologist, but that he should be familiar enough with the technic of roentgenology to know whether the roentgenologist has used the best possible positions in which to bring out the sought for field; whether a sufficient number of plates have been made to view the field from all angles, and whether the plates have been overexposed so as to burn out the fine lines and shadows so necessary for the diagnosis of an early or a hyperplastic sinusitis.

I believe that Dr. Law's three capital C's—Co-operation, Consultation and Co-ordination—are essential if the rhinologist hopes to obtain the greatest amount of information from the roentgenograms. By sending all of his cases to one roentgenologist with whose technic he is familiar, he will always receive a similar set of films, and each new set will be like greeting an old friend as to number, position and density of the films. You will seldom find a roentgenologist that will make a definite sinus diagnosis from the films taken by another roentgenologist, so how can the rhinologist hope to correctly interpret films if he sends his patients to first one and then another roentgenologist?

My brother[2] used the X-ray as an aid to his clinical diagnosis, and if the films disagreed with his findings, especially in the ethmoid area, he was prone to disregard the film, as nearly all roentgenograms show more or less clouding or disease of this area. Whether this is due to the failure of the rays to penetrate the superimposed bone to give a clear-cut outline of the ethmoid cells, or whether the cells always contain a thickened membrane, I do not know. But I do know that my films showing a clean-cut, healthy ethmoid area are as rare as that rainy day in California.

Is it possible to have one or more ethmoid cells filled with a hyperplastic lining membrane (the patient complaining of a se-

rous or seropustular discharge and other symptoms) without demonstrable change of the mucosa around the middle turbinate? I believe that such a condition may, in fact ofttimes does exist for a variable length of time, during which time a clinical diagnosis is difficult or impossible to make, and such a condition can only be brought to light by the roentgenogram.

The same may hold true for the other sinuses, and I am quite sure that many maxillaries with thickened hyperplastic membranes, with a nocturnal discharge or drainage, would go undiagnosed if it were not for lipiodol, or its equivalent, and the X-ray film.

They say a picture does not lie, and yet how many perfect likenesses are there in a dozen poses taken by an expert photographer? Could not this be true in the X-ray film? I sadly fear, if we were to extirpate all of the ethmoid cells that show clouding or softening of the cell walls in the roentgenogram, few of our patients would have a complete ethmoid, and yet it is remarkable to note the number of physicians, especially internists, who place absolute dependence upon the X-ray diagnosis, especially when the roentgenologist reports no evidence of sinusitis.

I am sure that Dr. Widmann will show you a number of slides of errors in X-ray diagnosis. One slide will show a maxillary reported as filled with polyps, which revealed at operation—forty-eight hours later—only a chronic thickening of the lining membrane. Another slide will show a negative maxillary, which, after lipiodol injection, demonstrated a thickened diseased membrane, proven at operation to be correct. The third slide will show two reported absent frontal sinuses that disclosed a massive disease at operation, etc., but I shall depend upon Dr. Widmann to prove to you the importance of a clinical diagnosis with a check on your diagnosis by the roentgenologist's report.

I shall close by citing two cases—one an error of the X-ray films, the other a clinical error, each corrected by consultation. It would be like carrying coals to Newcastle to go into any of the details of making a clinical diagnosis before the members of the Triological, so I shall merely give a summary of the points of interest in the clinical versus the X-ray diagnosis.

Mrs. D., age 31, referred for intermittent sick headaches and postnasal discharge.

Summary of clinical examination:

Septum deflected to right.

Spheno-ethmoid area: Mucosa swollen and congested with a hypersecretion of a thick tenacious mucus. No evidence of polyposis.

Return washings from left maxillary showed thickened pinkish saline solution.

Tonsils deeply buried cryptic type, showing infection.

Summary of X-ray report:

"1. Frontal and sphenoid sinuses clear.

"2. Extensive ethmoid disease by way of hyperplasia. Aeration space on right side still fairly well maintained. Hyperplastic process with moderate exudative accumulation causing considerable interference with aeration capacity of left side.

"3. Maxillary sinuses clear in first films. Lipiodol filling shows evidence of moderate hyperplasia of a chronic infection of the left side.

"4. Deflection of nasal septum to right."

You are probably saying to yourselves, "What is Skillern driving at? The X-ray report confirms his clinical findings." But wait, let me read you Dr. John Kolmer's laboratory report, which I consider as part of the clinical findings. The cultures were taken in the office in the following manner: After thorough shrinkage, the nasal mucosa was washed with sterile normal saline solution. A cannula was passed into the sphenoids, and they were filled with sterile saline and the fluid withdrawn. The maxillaries were punctured beneath the inferior turbinate with a Lichtwitz needle, filled, and the fluid withdrawn. The nasal mucosa was again irrigated and a platinum loop culture taken from the fronto-ethmoid and spheno-ethmoid areas.

DR. KOLMER'S REPORT.

"Right sphenoid—Nonhemolytic streptococci and staphylococcus aureus.

"Left sphenoid—Nonhemolytic streptococci in pure culture.

"Right maxillary—Pure culture of staphylococcus aureus.

"Left maxillary—Mixed growth of hemolytic and nonhemolytic streptococci.

"Posterior ethmoid area—Nonhemolytic streptococci along with staphylococcus aureus and albus.

"Anterior ethmoid area—Hemolytic and nonhemolytic streptococci and staphylococcus albus."

This report, along with her history and symptoms, shows all of these sinuses to be infected, and yet the X-ray reported only left ethmoidal block with moderate hyperplasia of right ethmoid and left antrum. Another point of real interest is the different organisms, some in pure culture, infecting the different sinuses. A submucous resection and tonsillectomy have given the patient some relief, although she still complains of postnasal discharge and occasional headache. To date there has not been any polypoid changes in the mucosa, but the discharge has changed into a pustular one. I shall await with interest the laboratory report of the cultures taken at operation from the opened sinuses.

The second case of clinical error corrected by the roentgenogram is of even greater interest.

Dr. Peter referred the case to the Skillern service of the Graduate Hospital with the following history: Female, age 14 years. Was in apparent perfect health until struck over the left frontal sinus by the fist of her three-year-old brother. Four days later eye began swelling. On the tenth day abscess pointed in center of upper lid.

Examination: No tenderness or swelling over superciliary area. No tenderness at inner canthus of either eye when pressure was made upward on the floor of the frontal sinuses. Intranasal mucosa appeared normal. No discharge, even after middle turbinate was infracted against septum and cocain and adrenalin applied around the nasofrontal duct.

The X-ray film demonstrated extensive involvement of the left frontal sinus which Dr. Pfahler diagnosed as malignancy or acute sinusitis.

Without manifestations of intranasal sinus symptoms and as the patient was up and about the ward in apparent good health, excepting the small abscess in the upper eyelid, neither Dr. Coates nor I felt justified in opening the frontal sinus.

After five days of study, I finally found a small fistula in the supraorbital wall leading directly into the frontal sinus. At operation the sinus was found to be filled with infected granular tissue, the entire lining mucosa was loosened from the bone, and the ostium completely occluded by this mass of tissue. Ten mm. to the outer side of the ostium a fistula 2 x 5 mm. was found leading directly into the orbital cavity. When the floor was curetted, the inner third of the supraorbital rim came away as a sequestrum, showing the great amount of bone involvement, and yet there were no intranasal manifestations of this osteomyelitic process which was going on directly above the nasal opening of the ostium. In fact, after the necrotic material was removed the mucosa of the nasofrontal duct appeared to be in a healthy state.

Two years ago Babbitt[3] reported a similar case before this society as orbital cellulitis with a breaking down of the orbital rim. He recommended early intranasal surgery.

If the roentgenogram had not shown this frontal sinus involvement, I fear the child would not have been operated upon until the symptoms of a brain abscess had occurred, which was merely a matter of time, as the posterior bony wall showed several necrotic areas requiring curettement down to the inner table.

The above case proves the necessity of using every available means of arriving at a diagnosis, consequently instead of the usual clinical versus the X-ray diagnosis, the roentgenogram should be a part of every clinical diagnosis of sinusitis.

The pre-operative roentgenogram should be a part of every sinus chart as routinely as the laboratory report. Whenever the roentgenologist is called upon to find a possible hidden focus of infection in the nose, he should, if in any doubt of his diagnosis, recommend in his report a complete clinical examination by a rhinologist.

SUMMARY.

1. One roentgenologist should be the rule for each rhinologist.
2. The rhinologist should be able to interpret the roentgenograms and use them as an adjunct in his diagnosis.
3. The roentgenologist's report should be used as a check on the rhinologist's diagnosis.

4. A consultation should be held on any point of disagreement in the diagnosis of sinusitis.

5. A negative roentgenogram should never be accepted as a diagnosis without a clinical examination.

6. Excepting in rare instances, an operation is not justifiable on X-ray evidence alone.

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REFERENCES.

1. Law, Frederick M.: Interpreting Sinus Roentgenograms. Transactions American Laryngological, Rhinological and Otological Society, 1931, Vol. 37, pp. 335-338.

2. Skillern, Ross Hall: The Present Status of Skiagraphic Interpretations as an Adjunct in the Diagnosis of Catarrhal Affections of the Accessory Sinuses. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, September, 1922.

3. Babbitt, James A.: The Diagnostic Problems in Orbital Cellulitis. Transactions American Laryngological, Rhinological and Otological Society, 1930, Vol. 35, pp. 225-237.

LXXXV.

IMPLANTS IN NASAL DEFORMITIES: HISTORY AND USES.

HARRY L. POLLOCK, M. D.,

CHICAGO.

That part of the symposium assigned to me deals with the history and uses of implants in connection with nasal deformities. Celsus was the first and only one of the ancients to mention plastic surgery. He wrote entirely on plastics of the nose, lips and ears and did not go into general surgery. None of the other noted Greek, Roman or Arabian physicians seemed to know anything of this subject, for nowhere is mention made of this topic. Late in the fifteenth century Taglizcozzi, professor of anatomy and medicine in Bologna, published a lengthy treatise on plastic surgery, which gave real inspiration to contemporary surgeons, and he is now considered the real founder of plastic surgery. Drawing nearer our own time, Graefe, in 1818, was the first of our modern surgeons to devote his time and energy to plastic work, and he highly recommended the Italian method, while Diffenbach, by his life's work and interest, gave the greatest impetus to modern plastics.

The earliest record I am able to find on the use of implants was that published by Von Langenbeck in 1859, entitled "Beiträge zur Osteoplastik," which was followed, in 1861, by Ollier of Paris in an article entitled "Bony and Periosteal Transplants in Man." These publications were soon followed by the works of Franz and Fritz Koenig, Von Hacker, Schimmelbusch and Israel and many others up to the present day. All authors up to the beginning of the present century employed homogeneous substances, namely, bone and cartilage, in plastics of the nose, while heterogeneous substances, such as gold, silver, vulcanite, etc., were employed only in cranioplasty. Since the beginning of the twentieth century three types of heterogeneous materials have been employed by surgeons in plastics of the nose, namely, paraffin,

celluloid and ivory. R. Gersuny, in *Die Zeitschrift für Ohrenheilkunde*, in 1900, published a paper entitled "A New Subcutaneous Prothesis," describing the use of vaselin. He tells of a patient whose testicle he was obliged to remove because of tuberculosis. Returning to him a year later, seeking the Doctor's help in avoiding ridicule to which his call to army service would subject him, Gersuny injected vaselin into the scrotum with a perfect cosmetic result. Later, he used vaselin in cases of cleft palate by injecting it into the uvula and posterior pharyngeal wall. In cases of saddle nose he used it successfully and highly recommended this method. Following Gersuny's article, paraffin was substituted for vaselin and was employed very extensively until the evil after-effects became known, when it was discontinued by all ethical surgeons. We have all seen some of the bad results from its use. Dr. Joseph C. Beck, in his contribution on plastic surgery to Loeb's "Operative Surgery of the Nose, Throat and Ear," has enumerated twenty-two complications following its use. There is no question that all these difficulties and dangers are authentic and every one may occur in the practice of a plastic surgeon where paraffin is used to a great degree. Another danger not specifically enumerated is the formation of a paraffinoma which we class as malignant but not in the sense of a carcinoma or sarcoma. There is a gradual infiltration of connective tissue into adjacent structures as, for instance, when injected subcutaneously for a depressed saddle nose, the paraffinoma attacks the orbital cavity and may cause serious injury to these structures. As example, I cite the following case: Miss N., a young lady, twenty years old, consulted an ethical rhinologist regarding the appearance of her nose. She was advised to refrain from any interference, as the nose was perfect. Notwithstanding, she consulted an advertising charlatan, who immediately injected paraffin subcutaneously. For a short time she was apparently pleased with the result, but within a year there was noticeable a reddening and irregular surface at the site of injection. This irregular red mass gradually spread over the forehead into her cheeks and into the orbital cavity, accompanied by severe pains over the entire region. With this picture she came to us for relief, both for pain as well as for cosmetic reasons. The overlying skin was infiltrated

and so firmly connected with the paraffin that it became necessary to remove it with the tumor. Large masses of paraffin were removed from the whole region, with the result that while the pain was relieved the appearance of the area was unsightly and depressing. Subsequently, a flap from the arm was obtained which covered the defect, but it was white and has ever remained so, while the adjacent skin is red. Two attempts to elevate the depressed skin flap, one by implantation of bone, the other of ivory, resulted in failure, as the tension was too great, due to the fact that we were dealing with too much scar tissue.

Celluloid has been used but very little, for in practically every case it has sloughed out.

Ivory, the third substance, has been used by European surgeons for a number of years, but it is only comparatively recently that its use has been employed in America as a framework in plastic surgery about the face. Jacques Joseph, in *Deutsche Medicinische Wochenschrift,* in 1919, states that inasmuch as bone transplants often become absorbed, he has substituted ivory implants in the treatment of saddle noses, but at this time he is not prepared to give any data. However, in his recent textbook, he makes the following statement: "For the past ten years, following the experience of Von Glück who employed ivory for bony defects of the jaws and extremities, I have used ivory in the correction of saddle nose and total rhinoplasty and, as a rule, with very good results."

Bone and cartilage have been employed almost universally, the bone being obtained from either the tibia, rib or finger and the cartilage from the costal cartilage, the ear, or occasionally from the nasal septum. From my personal experience I consider ivory preferable to either bone or cartilage. Ivory as obtained from the tusks of elephants is similar in its construction to bone. I find it preferable to bone for the following reasons: (1) Ease of obtaining implant. If bone is employed it must be obtained from the tibia or rib, causing the patient to undergo a double operation, one for securing the implant, and the second, the plastic operation. When the implant is obtained from the tibia there is, first, a risk of infection with long continued suppuration and the danger of an osteomyelitis, and second, continued pain in the region of the

wound, and third, the possibility of spontaneous fractures. Cases of spontaneous fractures have been reported, even if only a small portion of the crest has been removed. If the implant is taken from the costal cartilage or rib, there is risk of infection of the rib wound with a resultant osteomyelitis of the rib or sternum, or empyema with a prolonged convalescence. If the cartilage is obtained from the ear there is a risk of infection, resulting in a chondritis or perichondritis, thereby causing a deformity of the ear. If the cartilage is taken from the septum and infection occurs, a septal abscess with all its evil after effects may follow. (2) Exact shaping of implant, (a) in shaping or bending implant, if bone is employed, there is the risk of breaking and spoiling its usefulness; (b) the difficulty in obtaining a piece of bone or cartilage of the desired size, as frequently the implant is too small and an additional piece is necessary. My experience has been that a single implant gives better results than where numerous pieces are used. When using ivory, it is possible to measure the size that is needed with a great deal of accuracy, though should it prove too large at the time of operation, it may then and there be reduced to the desired size. I always have many pieces sterile, ready for use, and it is an easy matter to select the required one. The edges of the bone transplants are often rough and sharp, sometimes causing them to penetrate the overlying skin. This complication does not occur with the use of ivory, as its edges can be made as smooth and round as desired. (3) In sterilization, ivory, unlike bone or cartilage, can be boiled and put through the strongest disinfectants without affecting its usefulness. (4) Absorption of implant. There is little or no chance of the ivory implants being absorbed, for now, more than eleven years since these transplants have been used, there is no record of even the earliest cases showing a disappearance of the ivory. On the other hand, there has been and still is much discussion as to the absorbability of bone and cartilage. Most authorities agree that if bone with its periosteum, or cartilage with its perichondrium, be used as implants and brought into direct contact with living bone, there is little danger of its being absorbed. In using these tissues for plastic work about the face, it is often necessary to bury them in soft structures and it is then that they are likely to become absorbed

or degenerate into fibrous tissue. Again, it has been observed that occasionally they hypertrophy, especially the cartilage, thereby causing a deformity.

The disadvantages of ivory are twofold. First, it is a foreign material and does not grow or live like bone or cartilage and become an integral part of the body. It has been shown that it eventually becomes encapsulated by a firm, fibrous capsule just as other foreign bodies, such as bullets, pieces of steel, etc., do. Owing to the fact that it does not become an integral part of the body there is a greater likelihood of its becoming displaced, which condition has been occasionally observed. At present I perforate the ivory implants with the hope that the connective tissue will grow through the small openings, holding them firmly in proper position. Second, the danger of the implant sloughing out must be considered. It is now over seven years since I first began to use ivory implants almost exclusively, and thus far none has sloughed out except in those cases where primary infection occurred.

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LXXXVI.

NOISE IN INDUSTRY: ITS EFFECT ON THE HEARING AND ON GENERAL HEALTH (A REVIEW OF THE LITERATURE).

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In considering this subject it may be advisable first to define the term "noise." Dennis[6] quotes several definitions, starting with the facetious statement of an elderly man that "Noise is anything that I don't want to hear." Webster says, "Noise is sound without agreeable or musical quality," and Dennis speaks of noise as "Sound produced by the irregular or unperiodical vibrations of a body of air." Gilbert[9] defines noise as "the rapid and irregular succession of different auditory sensations."

Spooner[22] says that the word noise is in all probability derived from the Latin "noxa," hurt; "nocere," to hurt. Whoever introduced the word "noise" into our language must certainly have had a sounder knowledge of acoustics than many business men and engineers today. Funk and Wagnalls say, "Noise—a sound of any kind, but especially of a confused or disagreeable kind. In acoustics, the confused sound obtained by the discordant mingling of a number of distinct vibrations," and suggest the derivation from the Latin "nausea."

All of these definitions suggest discomfort, and discomfort increased beyond certain limits of degree or time usually leads to bodily harm.

It has long been recognized that extremely loud explosive noises may be the cause of sudden deafness, often due to perforation of the ear drum when too sharply impressed with the violent vibration. This condition is at times found in gunners and bell ringers, but we wish to consider deafness as an occupational condition separate from deafness due to explosive trauma. It is well known that riveters and boilermakers are deafened, in some instances markedly, from the constant din, and in rare cases the

circulation through the brain is affected, so that giddiness and nausea follow. These phenomena are mostly observed in the early career of the worker; they are diminished when the sensibility of the ear to the persistent sound is decreased.

Spooner[22] says that most middle-aged persons living and working in big cities are slightly deaf, due to the roar and rattle of traffic, motor busses, taxis, engines, sirens, telephones and typewriters. This would indicate that it is not only the extremes of noise that can harm.

Blake, in Kober and Hayhurst,[2] says: "Under modern processes of manufacture the majority of the trades are conducted through the intervention of machinery, . . . with resultant introduction of a prejudicial factor (noise), especially and continuously invading the organ of hearing, and even in some instances affecting that other portion of the internal ear which is a peripheral organ of equilibration, it being clinically observable that high grades of impairment of hearing are more common among those metal workers whose day labor confines them to intense sounds made up largely of overtones of high pitch, than among the workers in noises of low pitch."

Gottstein and Kayser[10] found that of 75 smiths and machinists, 40 per cent were definitely hard of hearing, and only 39 per cent had normal hearing. Holt[13] found only 15 per cent of 40 coppersmiths with normal hearing. Barr[1] found normal hearing in only 9⅓ per cent of 100 boilermakers, while Hebermann[12] reports that of 31 boilermakers, none had normal hearing.

Layet,[14] according to Gilbert, says that coppersmiths, sheet-iron workers, blacksmiths and coopers are always hard of hearing and become progressively more so. Roosa[20] says that workers in a noisy atmosphere develop a true nerve lesion, increasingly so, according to Moure,[15] if the noise is in small, closed spaces, as in boilers. Observations on railroad employees by Boval[3] show that deafness among engine drivers and stokers increases with age and years of service. Roepke[18] reports deafness among spinners and weavers, and Coosemans[5] among beetlers.

Blake[2] says: "The consensus of opinion is to the effect that the degree of impairment of hearing in all these cases has a definite relationship to the duration and intensive character of the opera-

tive sound. Both ears were usually tested, but more particularly that which was habitually directed toward the sound source. Subjective noises were almost an invariable accompaniment of the impairment of hearing, but dizziness or vertigo was present only in the more advanced cases, where the upper tone limit in hearing was notably decreased. Dizziness is also more likely to be an accompanying symptom in mill and factory operatives, when to the noise of the machinery there is added a sensible vibration."

The affection of hearing incident to exposure to loud sounds of high pitch is usually thought to be one of the inner ear solely;[2] but Holt[13] thinks that changes in the middle ear and transmitting apparatus are mainly the cause of the impairment. Gradenigo[11] distinguishes three types: labyrinthitis without middle ear implication, chronic catarrhal otitis media with labyrinthitis, and chronic advanced middle ear disease with implication of the labyrinth and vertigo; while Roepke[18] is of the opinion that in the majority of cases both the middle ear and the labyrinth are involved.

The principal channel through which the internal ear is subjected to injury from aerially transmitted sound waves of considerable amplitude, seems to be the sound transmitting apparatus of the middle ear. This idea is supported by the fact that it is not the tones of medium low pitch, most readily transmitted by bone conduction, which are the operative factors in the majority of cases of occupational origin, but those of comparatively high pitch which find their access to the internal ear through the sound transmitting apparatus only, the short sound wave impulses being almost immediately lost in the soft tissues.[2]

Moure[15] quotes Siebenmann[21] to the effect that pure sounds produce lesions in definite areas of the organ of Corti.

Castex[4] called attention to vibration, as in engine drivers and stokers, as acting on the labyrinth and causing sclerosis, and he thought that air transmission had little effect. Peyser[16] says:

"1. Continual but moderate noise, exclusively transmitted by air, has little or no action on the organ of hearing.

"2. Short but intense and shrill sounds, especially when repeated, injure the organ of hearing in a temporary or permanent way.

"3. Simultaneous conduction of noise by the air and by the bony skeleton, and above all, by the vibration of the ground, affects hearing finally and seriously."

Blake[2] says that a number of reports on animal experiments show very definitely that high pitched tones, and metallic sounds of lower pitch under resonance conditions simulating those of the trades as exhibited in factory operatives, machinists and boilermakers, may produce degenerative changes in the organ of Corti, beginning in the nucleated ciliated cells, progressing to the neuron, and then secondarily attacking the vibrating mechanism and extending to the contiguous membranous labyrinth. These degenerative processes were found to bear a measure of relationship to the intensity and duration of application of the invasive tone.

Rodger[19] found that the predominant noises to which the individual had been exposed determined the site of the initial lesion of the ear, and that for a considerable time the depreciation of hearing is mainly for sounds of a pitch corresponding to those noises. Later on there develops a marked loss of hearing for high tones, owing to the greater vulnerability of the lower part of the cochlear canal.

In all of the trades which entail more or less continuous exposure to heated air laden with dust, or foul with toxic gases, the effect on the ear is secondary to that induced in the mucous membrane of the nose and nasopharynx, varying according to the degree and character of the irritation or infection, from a non-suppurative thickening of the soft tissues of the middle ear to a suppurative process more or less destructive in its course. When in addition there is the loud noise of continuously operative machinery, the cycle of injurious effects, so far as the auditory transmitting and perceptive apparatus is concerned, is complete, and the welfare work of the trades will not have become adequate to its purpose until it has eliminated dust and noise as essential effects.

Dietrich[7] emphasizes the harm of industrial noise-deafness and dust-deafness as hazards of noisy industries. Industrial noise may not only be harmful to those workers in the noisy operations, but may be almost as deafening and more nerve racking to those in the neighborhood.

In a classification of primary noises Spooner[22] distinguishes between:

A. Road and rail traffic noises.
B. Street and house noises.
C. Industrial noises.

Subdividing the last group he lists:

1. Industrial noises due to machines.
   a. To prime movers. (Explosive types require special foundations and mufflers.)
   b. To transmission machinery: Poor belt joints, links, poor balance, defective bearings and vibrant mountings.
   c. To working machines (due to poor balance, vibrant mountings, etc.).
2. Industrial and nonmachine noises.
   a. To impact tools (at times eliminatable by substituting pressure for impact, as in hydraulic riveting).
   b. To movement of materials (often carelessness).

Among methods of lessening such noises may be listed shock absorbers on machines, rubber tires on trucks, rubber heels, rubber or cork treads, wood block floors to avoid vibration, proper proportioning of rooms so that sound reflections will occur at regular and commensurable intervals (the best proportions being length equal to height plus width, or height one, length three and width two), cellular wall construction, and discontinuous girders to lessen sound transmission. Manufacturers should realize that noise prevention and reduction is a paying proposition, increasing output in quality and quantity.

In addition to the effect on the hearing, "noise in its cumulative effects probably represents one of the most active causes of fatigue and therefore one of our greatest wastes."[22]

Scientific design from the standpoint of noise substitutes pressure for impact wherever possible. The hallmark of good design is silent running. In addition to accurate construction there must be efficient lubrication and efficient maintenance.

Spooner says[22] that apart from muscular fatigue the output of the worker is primarily affected by the quality of the air he works in, the efficiency of the lighting arrangements, and the amount

of noise in the vicinity. Very little has been done in any country to protect the worker against the fatiguing effects of noise. Most men soon become used to noise of any kind and do not feel the nerve strain due to it, nor are they aware of its fatiguing effects. The development of aural tolerance does not preclude harm.

"A period spent in the midst of working machines and mechanical operations, causing deafening and strident sounds of a very wide range, and shocks to the auditory nerves, produces a feeling of sensory fatigue. Although this fatigue attacks primarily a single organ, it little by little extends to the whole of the nervous system, leading in some cases to such a feeling of weariness that it may impair for work in a greater degree than severe muscular fatigue."[22]

Coosemans says[5] that in order for a noisy trade to be harmful it is necessary either—

"a. That the worker should be predisposed to affections of the ear by the presence of lesions in the nose or pharynx,

"b. That the noise should be intermittent,

"c. That it should be of a relatively high tone."

Gilbert[9] recommends exclusion of the obviously hard-of-hearing from noisy trades, and periodic ear tests of workers in noisy trades. The wearing of ear plugs, as of wax and wool, the lessening of floor vibration, and the lessening of noise production or varying of tone—lower for higher.

H. M. Vernon says[23] that the fatiguing effect of noise is chiefly due to its influence on the faculty of attention. Noise distracts attention and therefore necessitates a more intense mental application.

The noisy operations of industry may be listed as spinning, stamping, riveting, hammering and falling of materials.

Dietrich[7] lists among the evidences of the harmful effects of intense or continuous noise on the otherwise healthy: Increase and irregularity in pulse rate, palpitation, tinnitus, headache, irritability, fatigue and mental disturbance. Disturbances of circulation and nervous conditions can be definitely increased by noise.

Dennis,[6] reporting the results of the New York survey and study of municipal and industrial noises, states the following:

Studies on basal metabolism of typists showed a rate increase of 19 per cent in a noisy room, with increased heart rate and blood pressure.

A 12 per cent increase in output of office workers following a reduction of the noise level from 45 to 35 decibels.

A 4.3 per cent increase of output of skilled typists from the reduction of the noise level from 50 to 40 decibels.

A 42 per cent reduction in errors in the telephone room of a telegraph company with a 3 per cent reduction in cost, from a reduction of the noise level from 50 to 35 decibels.

A pamphlet distributed by the Deutsche Gesellschaft für Gewerbehygiene,[8] which has formed a special committee for combating impairment of hearing due to industrial noise, very well sums up as follows what we have tried to express:

"Noise can impair hearing by its intensity as well as by its duration; noise is transmitted not only through air, but through the walls and floors of the working rooms. A single strong sound impression, such as a whistle, explosion or stroke, is especially injurious if it occurs suddenly and unnexpectedly.

"The noise injures the most sensitive and vital parts of the organ of hearing, namely, the auditory nerve endings in the cochlea.

"Not every human ear is injured by loud continual industrial noise. The greater the power of resistance of the organism, and the healthier the organ of hearing, the less harmful is the effect of the noise.

"In the case of impaired hearing the individual is at first incapable of perceiving high notes, as tinkling, whistling, and the notes of a violin. In cases of prolonged duration of injurious effects of noise, the degenerative process extends to the other auditory cells which serve for the perception of low tones.

"Impairment of hearing caused by continuous, moderate occupational noise develops gradually and is at first hardly noticed. In the beginning, many workers do not feel the influence of the noise of the workroom. Examination by an ear specialist often reveals the impairment of hearing in such persons who are not aware of their injury.

"Stopping the ears with plugs of cotton or of flax is not sufficient protection against noise of workrooms. The ears should be protected regularly with compact plugs made of gauze saturated with petrolatum or wax. If the floor vibrates, straw, cork or rubber soles or felt shoes may be helpful; for kneeling, felt mats may be used.

"Apprentices or young workers, if coming from families with hereditary ear troubles, should not work in noisy factories. If they suffer from disease of the ear they should consult an ear specialist before entering such employment. When beginning work in an especially noisy plant, an examination by an ear specialist is advisable. During employment the examination should be repeated from time to time.

"A worker who finds that a noisy occupation impairs his hearing or is detrimental to his general condition should choose different work.

"In all cases of beginning impairment of hearing, an ear specialist should be consulted. This should not be postponed until serious disturbances become noticeable, such as ringing in the ears or attacks of dizziness. The earlier treatment is begun, the better is the prospect for improvement or recovery.

"Endeavors that strive to limit industrial noise should be promoted and supported. They include the proper distribution of machines, and suitable contrivances for the suppression of noise. Relaxation in quiet surroundings when away from work is the most helpful means of preventing ear troubles."

BIBLIOGRAPHY.

1. Barr, John Stoddard: Enquiry into the Effects of Loud Sounds upon the Hearing of Boilermakers. Glasgow, 1886. Quoted by Roepke in (18).

2. Blake, in Kober and Hayhurst: Industrial Health. Blakiston, Philadelphia, 1924.

3. Boval, G.: Quoted by Gilbert in (9).

4. Castex, André: 1897. Quoted by Gilbert in (9.)

5. Coosemans, E.: L'Audition chez les Bettlers. Contribution à l'étude de la sudité professionelle. Transactions Sixth International Otological Congress, London, 1899.

6. Dennis, E. B.: Noise, Its Measurement and Control. N. Y. State J. M., 30, 10, 573-576, May 15, 1930.

7. Dietrich, E.: Geräuschbelästigung und gesundheitliche Lärmschädigung. Die med. Welt, 8, 283-284, Feb. 23, 1929.

8. Foreign News Letter (Berlin), Jour. Amer. Med. Assn., 92, 2119, June 22, 1929.

9. Gilbert, D. J.: Influence of Industrial Noises. Jour. Industr. Hygiene, 3, 264-275, 1921-22.

10. Gottstein and Kayser: Über die Gehörsverminderung bei Schlossern und Schmieden. Bresl. Ärtzl. Zeitsch., 1881, Nr. 18, S. 204.

11. Gradenigo, Guiseppe, in Schwartze's Handb. der Ohrenh., Bd. II, Seite 467.

12. Habermann, Johann: Über die Schwerhörigkeit der Kesselschmiede. Arch. d. Ohrenh., Bd. XXX, Seite 1, 1890-91.

13. Holt: Die Schwerhörigkeit der Kesselschmiede und das Hören in geräuschvoller Umgelbung. Transactions of Fifteenth American Otological Congress, Ges. Ref. Arch. f. Ohrenh., Bd. XX, Seite 62.

14. Layet: Sixth volume Encyclopédie d'Hygiène de Rochard. Quoted by Gilbert in (9).

15. Moure, E. J., and Cauzard: Quoted by Gilbert in (9).

16. Peyser, M. W.: Quoted by Spooner in (22).

17. Richardson, Sir Benjamin: Health and Occupation, London, 1879.

18. Roepke, Friedrich: Die Berufskrankheiten des Ohres und der oberen Luftwege. Wiesbaden, J. F. Bergman, 1902.

19. Rodger, Ritchie: Quoted by Blake in (2).

20. Roosa, D. B. St. J., and Douglass, B.: The Ear, Nose and Pharynx, Macmillan Co., 1905.

21. Siebenmann: Private communication to Moure and Cauzard (15), quoted by Gilbert in (9).

22. Spooner, H. J.: Health Problems Involved in Noise and Fatigue. The Nation's Health, 4.2, 91-95 and 4.3, 156-159, February and March, 1922.

23. Vernon, H. M.: Noise. Welfare Work, 2, 83-85, 1930. Abstracted in Journ. Industr. Hyg., 12, 10, 235.

LXXXVII.

FIVE PRELIMINARY NOTES ON NASAL FUNCTION.*

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The following observations are presented as preliminary reports because enough data have not accumulated at present writing to justify final conclusions being drawn. They have been made with the utmost care, however, and, it is felt, frequently enough to warrant their publication.

1. *Cilia.*—The direction of propagation of the ciliary waves observed in the nasal mucous membrane of rabbits and man appears to be opposite to that of the streaming. In other words, the wave propagation is in the direction of the recovery stroke and not the effective stroke of the cilia. It is unlikely that this is an optical illusion. The mucosa has been observed under various conditions of illumination and neither they nor the angle of observation alter the phenomenon. The possibility of a stroboscopic effect of one file of cilia superimposed upon the next was considered. The waves were recorded with a motion picture camera and an attempt was made to change the appearance of the wave by varying the camera speeds. This also had no effect on the phenomenon.

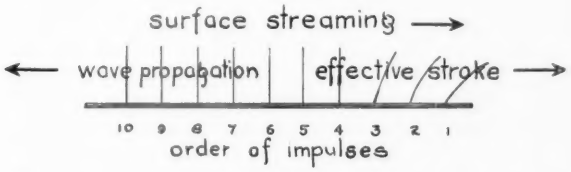

Fig. 1. Diagram to indicate the order of impulses, hence the direction of wave propagation as being opposite to the direction of the effective stroke which determines surface streaming.

*From the Department of Otolaryngology, Washington University School of Medicine, and the Oscar Johnson Institute, St. Louis.

2. *Cilia.*—A method was developed of obtaining a vertical view of the ciliated membrane in action and studying wave forms on the surface instead of in the usual profile. Portions of the rabbit's intranasal structures were removed en masse and placed upon a warm stage without separating the mucosa from the underlying bone. Under powerful lateral illumination, the cilia could not be seen owing to the glare of the light upon the mucus coat. The surface was therefore kept barely moist with physiologic sodium chloride solution and the ciliated epithelium was permitted to propel from its surface most of this coating of mucus. After this had occurred, the irrigation was stopped and the specimen allowed slowly to dry. During this process there occurred a period, usually less than a minute, when the ciliary wave motion could be observed, after which the drying put an end to it. Studies were made with the motion picture camera under various conditions of

Fig. 2. Diagram representing progress of waves. Wave begins at *A*, widens and deepens at *B* and disappears at *C*.

illumination and camera speeds to eliminate as much as possible the likelihood of optical errors. The following wave forms were noted:

The waves usually appear as small depressions or dimples, occasionally as short crescents; no wave front of any great length was seen. These "dimples" appear at first as shallow depressions, which rapidly deepen and widen and as rapidly reverse the process and disappear. During this cycle, the depression moves along the surface for a distance several times its own width (Fig. 2). All the dimple waves move in a general direction or rather along a definite path which does not vary; but this motion is not parallel even for a single microscopic field (Fig. 3).

The duration of the cycle is roughly one-fourth of a second.

The dimples succeeding one another may or may not recur in exactly the same location—that is to say, a second wave, involving a given group of cilia, may have its starting point slightly to the right or left or in advance of the starting point of its predecessor. Thus far it has not been possible to detect on these preparations whether the progress of the dimples is with or against the current of ciliary streaming.

3. *Cilia.*—Fragments of mucosa removed from sinuses at radical operation were immersed at once in physiologic sodium chloride solution at body temperature and examined for ciliary action. A large majority of specimens so examined revealed ciliary activity apparently normal in force, speed and distribution. From six to fifteen areas were examined in each case; as a rule, normal activity occurred in each area, although specimens were taken

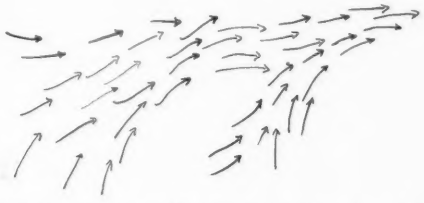

Fig. 3. Diagram indicating wave directions in a single microscopic field.

from the surface of extremely hypertrophied membranes, bathed in pus, removed from patients who had undoubtedly been infected for many years. At present, the noses examined number fourteen. In thirteen of these, ciliary action was found in each area examined but not always over the whole area. In one case, that of an old ethmoid infection with polyposis in which many operations had been done, no cilia were found anywhere.

Great care was not necessary in making these examinations to preserve ciliary activity. The action continued unabated from thirty minutes to three hours, with no other precaution than keeping the specimen moist with physiologic sodium chloride solution. In most cases no special warming was required; the ray from the arc lamp, filtered through a cooling chamber containing distilled water, supplied sufficient heat to the slide.

Fig. 4A. Two aluminum plates—vertical insulated from horizontal. Cigarette ash lying on horizontal plate.

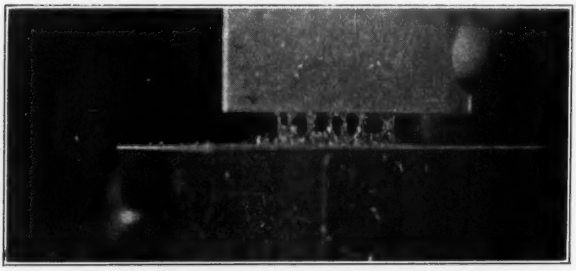

Fig. 4B. Same as above on touching upper plate after walking on carpet. Particles cling to upper plate.

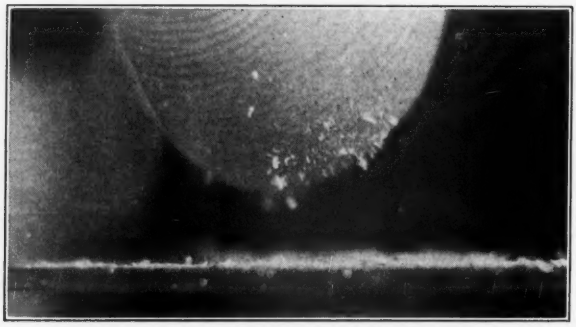

Fig. 4C. Particles lying on glass slide jump to finger a distance of .5 cm. or more.

The ciliated surfaces endure much abuse. They may be pressed between slide and cover glass, teased and torn apart until a few cells at a time can be observed without appreciably lessening ciliary activity. (Hilding has photographed individual cells upon which cilia were still active.)

4. *Cilia.*—When distilled water was substituted for the sodium chloride solution, the cilia quickly became cloudy and motion promptly ceased. This could not be re-established by returning to the salt solution.

When, on the other hand, the concentration of the salt solution was increased, approximately five times the normal salt content was tolerated before the action ceased. If the specimen was promptly washed with distilled water and the normal salt solution was again supplied, the ciliary motion recommenced and continued indefinitely (fifty minutes, when the experiment was stopped).

5. *Surface Charges.*—When the atmospheric humidity is low, especially indoors in winter after furnaces have been in use for some time, the friction of clothing and of shoes on carpets generates a positive charge on the body surface. This includes the surface of the nasal mucosa. This charge is sufficient to cause the prompt adherence of dust particles and other minute negatively charged bodies. This was demonstrated by preparing two aluminum plates at right angles to one another and separated about one-half inch by means of a bakelite rod. When cigarette ash was scattered upon the horizontal plate and the vertical plate was later touched during some slight motion producing friction on clothing or carpet, the ash immediately adhered to the edge of the overlapping plate. (Figs. 4A and 4B.)

These charges were recorded by means of a string galvanometer through an electrode in contact with the various surfaces in turn: face, nasal mucosa, mouth and pharynx. It was observed that any slight motion produced variations in the current, also that respiration did not have the effect of lowering the pharyngeal charge, the discharges remaining the same whether the patient breathed or not (Fig. 5).

It is suggested that this surface charge may be an important factor in removing bacteria, which are known to bear a negative

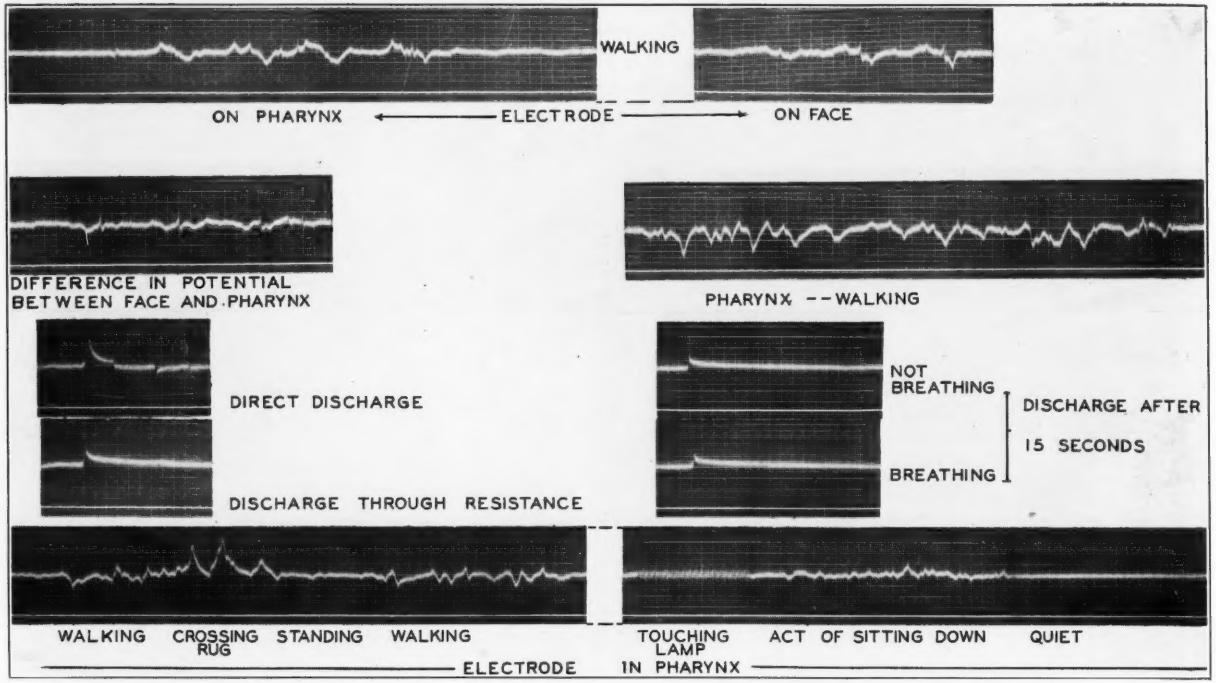

Fig. 5. Galvanometer records of surface charges induced by moving about a room.

surface charge, from the inspired air, segregating them in the anterior portions of the nose. (Linton and others have reported the postnasal spaces practically free of bacteria.)

Attempts have been made to verify this action of surface charges by observing the distribution of inhaled powders with the body surfaces at ground potential and under various charges. Thus far these have not been successful.

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LXXXVIII.

THE RED NASAL SEPTUM SYNDROME.*

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The upper respiratory tract presents a number of variations to the eye of the observer. It may present a variation in normal contour requiring surgical correction. Or it may present a variation in micro-organism infestation changed into micro-organism infection requiring local or general treatment or possibly an application of the surgical principles of ventilation and drainage. Then again it may present a variation in the color of its mucous membrane, these mucous membrane color changes serving as an index to the body chemistry of the individual being examined. It is this last variation that I wish to consider more at length at this time. These mucous membrane color changes present to the eye of the observer seven distinct patterns: One of these patterns deals with the degree of redness of the mucous membrane covering the turbinates, more particularly the inferior turbinate. Three deal with the degree of pallor of the mucous membrane covering the cartilaginous portion of the nasal septum, and three with the degree of redness of this same mucous membrane. These last three patterns, representing three degrees of redness—light red, dark red and crimson red—have associated with them a syndrome which is most prominent and well developed when the mucous membrane covering the cartilaginous portion of the nasal septum is crimson red in color. As to the factors underlying the production of the crimson septum and the syndrome, they seem to be three in number. In the order of their importance, they are first crossing the line of tolerance for acid ash producing food; second, long continued intake of medicinal acids, and third, fatigue due to excessive output of muscular energy. The trio usually met with consists of crossing of line of tolerance for acid

*Presented at meeting of Eastern Section of the American Laryngological, Rhinological and Otological Society, New York City, January 9, 1932.

ash producing food, crimson mucous membrane covering the cartilaginous portion of the nasal septum and the syndrome.

THE SYNDROME.

The syndrome may be described as follows: These individuals are tired all the time. The day's work, instead of being a joy, represents a round of duties to be gone through with. They are irritable when they do not mean to be. They are a bit hard to live with, when that is their least desire. They go about with a hair-trigger type of temperament, being easily thrown off balance by any unexpected situation arising during the day. While they participate in the three meals of the day, there is no zest for food. In the middle of the day, when they should be awake, they are drowsy. When bedtime arrives and they should be drowsy, they are most awake and at their best. Their night's rest brings them only a moderate amount of refreshment, coming to the beginning of the day's work a bit tired. They are subject to indefinite symptoms of short duration in various parts of the body. They are constipated, their skin is dry, their hair comes out a bit more than they wish. They shun their social duties, feeling that the day's work represents the extent of their endurance. All in all, they feel a long vacation of some sort should be indulged in order for them to feel right again.

PRE SYNDROME.

With the dark red mucous membrane covering the cartilaginous portion of the nasal septum, there is associated a pre syndrome which may be described as follows: These individuals, while able to do the day's work, realize that they have very little reserve energy. They are able to follow fairly well a definite daily routine, but experience difficulty in meeting a new situation which arises in the day's work. They find difficulty in surviving a short period of additional business or professional pressure or increased social duties. They prefer that their outside activities be mental instead of physical. They readily fall a prey to the infections but usually recover. These infections are generally of an acute nature.

INTERPRETATION OF SYNDROME.

These individuals have a low tolerance for acid ash producing foods, medicinal acids or excessive output of muscular energy.

Their metabolic process is apparently such that during metabolism a larger quantity of acids is formed than can easily be neutralized by the cell base, the intercellular fluids and blood plasma. In excreting this excess of acids resulting from metabolism fixed base is removed from the body with a resulting negative base balance. The crimson redness of the cartilaginous portion of the nasal septum enables one to suspect what is taking place in the body of the individual. The presence of the syndrome verifies this suspicion.

TREATMENT.

If the observations, deductions and conclusions have been correctly made, then a reduction in the intake of acid ash producing foods, the giving of an alkalin preparation with a view to replacing the base balance; the use of a catalytic agent which will excite the intermediary mechanism of the body to pick up more of the minerals from the digestive tract and an increase of the patient's fluid intake in order to balance the intercellular fluids ought to cause a disappearance of the syndrome and a reduction in the degree of redness of the mucous membrane covering the cartilaginous portion of the nasal septum. This they do. One month is generally required before the patient begins to notice a beginning disappearance of the syndrome. Three to six months are required before the nasal septum begins to show a decrease in the degree of redness of the mucous membrane covering the cartilaginous portion of the nasal septum.

To be more specific with reference to treatment, a number of requests are made of the patient. First he is given a food list showing acid ash producing foods in one column and alkalin ash producing foods in another. Until his syndrome has disappeared and until the degree of redness of the mucous membrane covering the cartilaginous portion of the nasal septum shows a satisfactory decrease, he is requested each meal to select three foods from the alkalin list to one food from the acid list. Following the disappearance of his syndrome, he is requested to select one food from the alkalin list to each food selected from the acid list. In case he notes a reappearance of the syndrome he is to return to the three to one ratio. The second request is that

he take an even teaspoonful of sodium bicarbonate in a glass of warm water at 10 a. m., 3 p. m., and 8 p. m. for three days. At the end of this three day period he is requested to take a glass of lemonade made from half a lemon, without sugar, at 10 a. m., 3 p. m. and 8 p. m., for three days. Then sodium bicarbonate is taken again at 10 a. m., 3 p. m. and 8 p. m. for one day and lemonade again for three days. He is to continue this rotation of three days of lemonade and one day of sodium bicarbonate until one month has passed. At the end of the month he is to continue lemonade once a day. The third request is that he take one teaspoonful of cod liver oil at the morning meal and again at the evening meal until he can sleep without morning fatigue. When this bodily state is reached he is requested to continue the cod liver oil once a day indefinitely in order that a fairly high vitamin intake may be maintained. The fourth request is to drink a glass of water every hour from the time of awakening until the evening meal. This increased water intake is continued until puffiness of the eyelids or bloating of the ankles is noticed. The object of the increased fluid intake is to balance the intercellular fluids of the body. When this bodily state is reached, the fluid intake is reduced to a glass of water every two or three hours during the day.

PRACTICAL VALUE.

By a study of the mucous membrane covering the cartilaginous portion of the nasal septum, you will be able to diagnose incipient or actual staleness in your patient. The presence of the pre syndrome or actual syndrome verifies your upper respiratory tract deductions. The presence of incipient or actual staleness in your patient is a handicap to securing results from your usual treatment, either local or general, for a given clinical condition. As a guide to pre-operative or post-operative treatment, the color of the mucous membrane covering the cartilaginous portion of the nasal septum assumes an added importance, for it points the way to a satisfactory or unsatisfactory body chemistry. The state of the body chemistry influences the length of the convalescence. I wish to report the following case as illustrating the relationship existing between the increased redness of the mucous membrane

covering the cartilaginous portion of the nasal septum and the syndrome which has been outlined.

Patient, age 23, attending college in Boston, preparing for social service work. This is second college year. Sent home from college by physician consulted because of impending nervous breakdown. Was advised to consult a throat specialist because of chronic sore throat. Came to office direct from train on arrival in Barre, March 16, 1931. Have known this girl from infancy. She has always been a strong, athletic type of girl. Able to swim long distances and engaged in all sports in which girls generally engage. Was surprised to see the loss of color in her face and lack of pep she manifested. An examination of her upper respiratory tract showed mucous membrane covering cartilaginous portion of the nasal septum to be crimson red. The anterior pillars showed this same degree of redness, and lymphatic bands posterior to each posterior tonsillar pillar were prominent and red. There was an absence of mucopurulent discharge in the nose and throat. The turbinates were normal in color and size. Both tonsils well removed at eight years of age. Sinuses transilluminated well. After an examination of her upper respiratory tract I outlined the syndrome as representing how she felt. She stated that represented the way she felt.

The interpretation of the upper respiratory tract examination may be outlined as follows: The redness of the septum and anterior pillars showed that the patient was selecting food more from acid producing food than alkalin producing foods. The absence of a mucopurulent discharge showed that she had not exceeded her tolerance for starches. The absence of a watery nasal discharge and increased redness and swelling of the turbinates showed she had not exceeded her tolerance for sweets. For treatment she was asked to take one teaspoonful of cod liver oil at the morning and evening meals in order that her daily vitamin intake might be increased. To use a sodium bicarbonate gargle after each meal. She was asked to give up all meat, cereals and bread and to eat two oranges each meal. She was put on the alkalin rotation of sodium bicarbonate and lemonade and was given a food list with the request that she select three foods from the list of alkalin producing foods to one from the list of

acid producing foods. She reported by phone two days later that she was feeling very much better. Five days following her visit to the office her mother phoned that her daughter had followed the directions I had written down and was feeling more like her old self again. The color had returned to her face, and she was planning to return to college. She returned to college and followed out the regime outlined to her. She finished the college year and worked all summer in charge of a Chamber of Commerce tourist station. Up to the present writing she has had no return of the syndrome. This case illustrates the rapid progress it is possible to make in correcting a nutritional disaster in a patient whose nutritional disaster has not progressed too far and whose body has the ability to right itself if given a little bit of help.

QUARRY BANK BLDG.

LXXXIX.

RELATIONSHIP OF NASAL SINUS INFECTION TO SYDENHAM'S CHOREA.*

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Chorea of childhood, or St. Vitus dance, was first described by Sydenham in 1686. It is defined as a disease of the central nervous system, characterized by irregular involuntary muscle contractions, which may continue during rest, but usually cease in sleep and are increased by excitement and emotion.

Recurring chorea interferes greatly with development and education, and is a serious handicap to the growing child. It is a fairly well established fact that the disease is associated with or follows infection. The presence of an existing focus of infection in these patients should be established as early as possible.

The female has the disease more frequently than the male in the proportion of at least three to one. The breast fed infant is said to be immune. The largest percentage of patients are from 9 to 11 years old, 75 per cent from 6 to 15 years old and about 13 per cent of patients from 16 to 20 years.

The mortality in chorea is about 3 per cent, with death usually due to endocarditis or pericarditis. Osler says there is no other disease in which endocarditis is so constantly found postmortem as chorea. Osler states also that the primary heart trouble in chorea is in most cases endocarditis and that in most of these cases some trouble with a few physical signs will continue throughout life.

Some writers of fifty years ago noted the association of nasal infection with this disease, the site of the infection supposedly being in the nasopharynx.

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*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Western Section meeting, Los Angeles, January 29-30, 1932.

Stratton[1] (1885) suggested the hypothesis that chorea was due to a micro-organism which penetrates through a slight break in the continuity between the nasal and pharyngeal mucosa.

Jacobi[2] (1886), under the title "Partial and Sometimes General Chorea from Naso-Pharyngeal Reflex," states: "The inspection of the nose reveals a congested membrane and thickening and hyperemia of the cavernous tissue, particularly of the lower concha. The nostrils or one of them are always narrow, the septum deviated; nasal catarrh is frequent."

de Ponthiere[3] (1910), under the title "The Nasopharyngeal Origin of Chorea," describes the frequent colds and states that for twelve years the author has had his attention drawn to the pathogenesis and treatment of chorea, and he must say, without hesitation, that he is convinced that much more frequently than is believed a rapid and permanent cure, and not a passing improvement, depends upon relief of this condition.

Diseased tonsils have for some time been considered a factor in Sydenham's chorea, but only in recent years has the relationship of nasal sinus infections been proven.

Cone,[4] in reviewing reports of cases of various systemic diseases of infancy and childhood associated with paranasal sinusitis, states: "Classified in the chorea group were six with sinus disease, two with chronic suppurative maxillary sinusitis and one with empyema. Two had in addition chronic suppurative ethmoiditis and sphenoiditis, one was recorded as paranasal sinus disease and one as chronic suppurative ethmoiditis bilateral. Two of the six cases had the cardiac manifestations of chorea."

Piper[5] mentions two cases cited by Jean. A girl, aged nine, had chorea of four months' duration; tonsils and adenoids had been removed two years previously. Patient had a secondary anemia and was ten pounds under weight. Pansinusitis was found to be the cause, with very thick pus in the maxillary antra. A girl, aged seven, had chorea with a moderate mitral insufficiency. The girl was under weight and had secondary anemia. Chronic ethmoiditis was diagnosed. There was apparent complete recovery for two months, when fever and cough began and acute endocarditis developed. The ethmoid infection, which had persisted,

was found to be the only possible focus of infection. Active treatment of the ethmoiditis was followed by rapid convalescence. Patient was in good health within six months.

Phelps[6] reports six cases of chorea with heart findings in a total of 168 cases of sinusitis in children. Mitchell[7] lists ten cases of chorea with paranasal sinuses as a focus of infection in his tabulation of "The condition for which infections were sought for." McCulloch[8] believes cardiac disease in chorea should be considered joint result of some focus of infection, as in nose, throat, or paranasal sinus. Minard[9] states: "For the ultimate cure of chorea, remove the primary cause, if discoverable. This will usually be found as a residual focus of infection in sinus, tonsils, etc." Harkness[10] reports a case, a boy, aged eight, whose choreic symptoms showed most marked and rapid improvement after draining the maxillary antra. Arbuckle[11] states: "A few cases of chorea of a severe type with heart involvement apparently have responded with unusual rapidity after treatment of suppurative disease of the nasal sinuses had been instituted."

Skillern,[12] in the section on nervous conditions, quotes the following: "Certain conditions, as neuralgias, forms of tic douloureux, neuritis, and the like, have been reported as being caused by a sinusitis, and in children the severe forms, such as chorea; in fact, Crane states that many children would be saved from acute rheumatism, with attending heart lesions and chorea, if the primary focus was early enough eliminated."

Marriott[13] says: "Sinus infections are usually observed in patients having true rheumatic endocarditis and other manifestations of chorea and articular rheumatism." In the author's experience, the clearing up of sinus infections has been more effective in preventing the recurrence of endocarditis, chorea and rheumatism than has the mere removal of tonsils and adenoids.

Kistner[14] mentions a case in which a very gross thickening in the antra of a child, nine years old, afflicted with chronic chorea for three years was found, operated on for tonsils and adenoids with no relief. Recovery followed exenteration of her sinuses.

Within the past ten years nasal sinus infection is mentioned as a focus of infection in chorea by fifteen other writers on this

subject. Reports indicate that many cases of chorea with involvement of the nasal sinuses respond very promptly to conservative treatment of the infected sinuses. Those cases with impending injury to the heart may demand a more prompt and certain removal of the infective focus.

At Doernbecher, children's hospital of the University of Oregon Medical School, Portland, Ore., in a total number of 5,328 cases admitted to the hospital for all causes, there were 52 cases of chorea; of these, 14, or about 25 per cent, had nasal sinus infection.

Following are the brief reports of four cases of chorea with antrum infection operated upon radically. Three were operated upon radically by request of the Chief of the Department of Medicine, to avoid injury to the heart, one because conservative treatment proved to be inadequate.

Case 1.—C. E., male, aged ten, entered hospital February 8th; an attack of chorea began three weeks previously. Patient unable to talk the past few days; severe irregular movements of face and limbs; pain in joints of right hand. Patient had had frequent colds in the head and had had one just previous to present onset of symptoms. Infected tonsils and bilateral antrum infection found. Conservative treatment of the antra was instituted and tonsils were removed. No improvement in condition followed, and a radical removal of the focus in the antra was requested by the medical staff.

March 1st a bilateral Caldwell-Luc operation. Pathologic report of exenterated membrane was chronic catarrhal sinusitis. Patient discharged from hospital April 6th; a letter from the mother, May 7th, reported child very well.

Case 2.—M. K., female, aged ten, entered the hospital February 6th. First attack of chorea four and one-half years previously, at which time tonsils were removed. Several recurrences since. Last attack in December; pains and swelling in left ankle and knee; good health aside from chorea. Examination of nose and throat disclosed thick postnasal discharge with bilateral antrum infection. Heart examination revealed endocarditis and radical operation was requested by the medical staff. February 27th, bilat-

eral Caldwell-Luc operation. Pathologic report: Polypoid hyperplastic membrane; hyperplastic sinusitis. Patient improved rapidly following operation. No complications. Discharged cured March 12th.

Case 3.—A. R., female, aged nine, entered hospital January 30th. Became acutely ill with chorea a month before. Pains in limbs and joints. Has had a cold constantly this winter. Septic tonsils and infected antra found. Tonsils removed February 13th. Antra did not improve with irrigations and drainage. Bilateral Caldwell-Luc operation February 26th. Pathologic report: Catarrhal sinusitis. Child improved steadily. Discharged March 8th. Returned to clinic for observation March 15th. Mother says child is cured. Marked systolic murmur still heard at apex of heart.

Case 4.—R. M., female, aged thirteen, readmitted to hospital January 14th, for a third attack of chorea within the past two years. Patient found to have chronic bilateral antrum infection. Treatment by irrigations instituted. During this treatment both ears abscessed, followed by right mastoiditis, which was operated upon February 10th. Following the mastoid operation choreic manifestations became very violent. Patient placed in restraint; could neither take nor retain food; lost weight rapidly; impossible to treat antra during two weeks. March 4th, general condition was found better and, as condition of antra had not improved with drainage, on March 15th bilateral Caldwell-Luc operation was performed. Both antra were found filled with thickened polypoid membrane. Pathologic diagnosis: Mucopurulent cystic and polypoid sinusitis. March 31st, patient discharged. May 16th, patient examined in outpatient department. Nose found to be clean. There had been no return of chorea. August 29th, patient reported and found in excellent health.

SUMMARY.

Chorea has been recognized for 250 years. The literature of fifty years ago suggests conditions in the nasopharynx as a cause of the disease, but only in the last few years has the relationship of nasal sinus conditions been recognized.

The number of chorea patients, with sinus infection, in Doernbacher Hospital has been 25 per cent of the total number of chorea patients.

It is probable that this percentage may be found to be higher as a more careful search for sinus involvement is made.

All writers have emphasized that rapid improvement, and often cure, follow prompt and active treatment of the sinus condition. Many urge a rapid elimination of the focus to save the heart.

The future may demonstrate that nasal sinus infections have more to do with this disease than is now supposed.

MAYER BLDG.

BIBLIOGRAPHY.

1. Ferrarini, C.: Contributo allo studio sull' etiologie della chorea di Syndenham (chorea post-influenza). Sperimentale. Communicz. e riv. Firenze, 1892, 231-241.

2. Jacobi, A.: Partial and Sometimes General Chorea Minor from Nasopharyngeal Reflex. Am. J. M. Sc., Phila., 1886, XCI, 517-522.

3. de Ponthiere (Charleroi), S. L.: (Translated by Macleod Yearsley.) The Nasopharyngeal Origin of Chorea. J. Laryngol., Rhinol., and Otol., Lond., 1910, XXV, 461-466.

4. Cone, A. J.: The Relationship of Acute and Chronic Paranasal Sinus Disease to Systemic Conditions in Infants and Young Children. Laryngoscope, St. Louis, 1927, XXXVII, 19-31.

5. Piper, A. S.: Focal Infection from Paranasal Sinuses. Journal Oklahoma Medical Association, 1928, XXI, 191-194.

6. Phelps, K. A.: Relation of Nasal Sinus Disease to General Medicine. Journal-Lancet, Minneap., 1929, XLIX, 424-427.

7. Mitchell, E. C.: Paranasal Sinus as Focus of Infection in Children. South. M. J., Birmingham, 1925, XVIII, 686-693.

8. McCulloch, H.: Cardiac Manifestations of Chorea. Methods of Treatment in Use at Ridge Farm. Trans. St. Louis Ped. Soc., Am. J. Dis. Child., 1925, 865.

9. Minard, E. L.: Etiology and Treatment of Chorea. J. Med. Soc. N. Jersey, Orange, 1930, XXVII, 219-222.

10. Harkness, G. F.: Nasal Sinus Disease in Infants and Young Children. Wisconsin M. J., 1928, XXVII, 112-118.

11. Arbuckle, M. F.: Systemic Manifestations of Suppurative Disease in Paranasal Sinuses. J. Am. M. Assn., Chicago, 1923, LXXXI, 741-744.

12. Skillern, R. H.: Focal Infections from Accessory Sinuses. J. Indiana M. Assn., Fort Wayne, 1925, XVIII, 8-12.

13. Marriott, McKim: Pediatric Aspects of Otolaryngology. Tr. Am. Laryngol. Assn., 1927, 470-476.

14. Kistner, F. B.: Pathology of Sinusitis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1929, XXXVIII, 977-994.

XC.

THE ETIOLOGY AND TREATMENT OF ORBITAL INFECTION.*

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In presenting this paper for your consideration I am approaching the subject from the standpoint of the ophthalmologist, as well as the rhinologist, having been trained in both specialties.

During my ophthalmic training (1912-1913), I saw orbital cellulitis and abscess treated on the whole inadequately by simple orbital incision and drainage, frequently without recourse to X-rays of the sinuses or consultation with the rhinologist. In recent years it has seemed that the rhinologists are in danger of making a similar mistake by ignoring the ophthalmologist. In my opinion, the condition is one frequently requiring the attention of both.

In reviewing the literature I find a scarcity of definite information on the subject. In the proper treatment of this disease an understanding of the anatomy of the orbit, with its relation to the adjacent parts, particularly the nasal accessory sinuses, is necessary. Following is a brief description of this:

The orbit is a quadrilateral pyramid—base forward. The base is closed by the anterior wall, continuous with the periosteum, the fascia tarso-orbitalis starting from the orbital margin and attached to both tarsi and to the external and internal canthal ligaments. Thus we have a pyramid shaped cavity whose bony walls, over about one-half of the entire area, are also the walls of the nasal accessory sinuses.

In this cavity is the eyeball, optic nerve, extrinsic muscles, ophthalmic artery, superior and inferior ophthalmic veins, communicating anteriorly with the veins of the face, as well as the sinuses, particularly through the anterior and posterior ethmoid

*Read before the American Laryngological Association, Atlantic City, June, 1931.

foramen, and posteriorly with the cavernous sinus, the motor nerves, ciliary ganglion and long ciliary nerves. Surrounding the eyeball proper from the cornea to the sheath of the optic nerve is Tenon's capsule. All the interspaces are filled with orbital fat. There are no lymphatic vessels or glands in the orbit.* Lying below the orbit is the antrum of Highmore. Mesially are the ethmoid and sphenoid sinuses, while above is the frontal with orbital prolongations and the orbital extension of the ethmoid cells.

Infections due to furuncles, carbuncles, injuries to the face, surgical trauma, are transmitted to the orbital cavity through the veins of the face. Tooth infections reach the orbit either underneath the periosteum or through the antrum. Sinus infections may extend to the orbit by direct continuity, either with or without necrosis of the bone or by a venous thrombosis or thrombophlebitis.

In the diagnosis of orbital infection it is well to bear in mind the various pathways mentioned, because the treatment depends somewhat on the source of the infection. A careful history and X-ray examination are the two most important factors in diagnosis. In almost all of the cases in this series that were secondary to sinus involvement the history of a recent cold or exanthema was very clear, and in 73 per cent of them there were positive X-ray findings. As shown in a small number of cases, X-ray examination of the teeth is important and should always be considered.

In infections secondary to a dacryocystitis, a history of tearing and antecedent infections of the tear sac will be of help. In those secondary to acute furuncles, carbuncles, etc., of the nose, lip and cheek or from infected wounds of the face, the source may seem fairly evident. Here, however, X-rays of the sinuses and teeth should be a part of the examination.

The question of differential diagnosis was so well covered by Dr. Babbitt in his paper last year at the meeting of the American Laryngological, Rhinological and Otological Society, that I have not attempted to go into this phase of the subject.

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*Fuchs' Textbook of Ophthalmology.

There are three distinct stages of the disease: First, edema of the lids; second, edema of the lids with exophthalmos, with or without limitation of motion; third, the same as the second but with evidence of actual abscess formation, pointing in upper or lower lid. Contrary to statements in various texts, I have not found the place of pointing to be indicative of the location of the place of entry of the infection. For example, the infected thrombus from a posterior ethmoid vein may finally point in the outer portion of the upper lid.

In describing the treatment of cases secondary to sinus infection, the method of procedure varies according to the stage or extent of infection present when the patient first comes under observation. Where possible, the patient should be hospitalized and X-rayed. If there is simple edema of the lids with history of recent upper respiratory infection, the conservative or expectant method should be adopted. This includes shrinking of the nasal mucous membrane with pledgets soaked in a 1 per cent cocain solution, two or three times a day, with warm albolene spray solution, either sprayed or dropped in the nose, in the interim. (I prefer never to use adrenalin in an acutely inflamed nose.) Following the shrinking of the mucous membrane, nasal irrigations of warm normal saline should be given and cold compresses should be applied externally.

If after twenty-four to forty-eight hours of this treatment there is no evidence of improvement, or if the condition has progressed, more active measures are necessary. In children, particularly with cloudy ethmoids and antra, removal of the anterior portion of the middle turbinate and opening of the ethmoid labyrinth, with possible intranasal antrotomy, will frequently be followed by rapid subsidence of symptoms and cure. In an adult it might be well, depending on the previous history and X-ray findings, to do a radical antrum instead of an intranasal.

If the case presents at first marked edema of the lids, exophthalmos with limitation of motion, radical measures are immediately advisable. This means that the infection has already invaded the orbit to the extent of setting up an infiltration of the orbital tissues. I have found the best results from an external ethmoid exenteration with removal of the floor of the frontal, if this is

involved, as well as a Caldwell-Luc type of antrum operation. The ethmoid incision should be left open and a rubber tissue drain inserted, as well as a light packing being placed in the nose. By this method of attack the orbital periosteal sheath can be palpated and inspected for areas of necrosis, and if none are found most cases will immediately begin to improve so that secondary closure of the wound can be done in a few days with no more subsequent deformity than if the wound were closed at the time of the original operation.

If, through neglect (or obstinacy on the part of the patient, as happened in one of my cases last year and one the year before), nothing is done until there is definite bony destruction in the ethmoids and frontals, as well as actual abscess formation in the orbit, it is necessary to do a modified Killian and at times an obliterating frontal sinus operation, besides radical surgery on the antra and drainage of the orbit.

In cases due to causes other than sinus infection, the treatment is similar in that it is directed toward removal of the cause. The orbit should not be entered until there is evidence of pointing or the edema and exophthalmos become so marked as to constitute a danger to vision.

The material for this paper is based upon a review of cases seen in private practice and case records of the Massachusetts Eye and Ear Infirmary since 1922, about sixty in all. In this study it is noticeable that orbital abscess is comparatively rare; most cases properly followed and treated do not get beyond the second stage. A majority of these abscesses are due to surgical trauma, i. e., by ill timed attempts to drain a condition not yet present. Approximately 75 per cent of orbital infections were secondary to infections in the nasal accessory sinuses, and the ethmoids were involved in all cases. Of these, about 82 per cent were in children, 7 per cent were directly due to abscessed teeth, 7 per cent to injury, and 13 per cent to miscellaneous causes.

The mortality was 5 per cent in this series, all in children under two years of age. One case showed at autopsy right pansinusitis, right empyema, right cavernous sinus thrombosis, right otitis media (J. J. B., 92422).

CONCLUSIONS.

1. A large majority (75 per cent) of orbital infections are due to acute infections of nasal accessory sinuses or acute flare-up of pre-existing chronic sinus disease. It is in this group that the most satisfactory results are obtained by early diagnosis, careful observation and proper treatment.

2. Next in importance as causative factors are tooth infections.

3. The orbit is very resistant to infection and real orbital abscess is comparatively rare. Many of them are due to too early surgical interference in the orbit instead of the treatment being directed to the pre-existing cause.

4. In very young children orbital infection is exceedingly dangerous.

The following three cases illustrate the results of proper treatment, delayed treatment and lack of treatment, from the onset of the disease:

Case 1.—J. F., aged 7 years. Past history: negative. Present illness: Tonsillitis with acute rhinitis and cervical adenitis. Seen in consultation at patient's home on February 22 and February 27, 1931, and advised removal to hospital. Admitted to hospital February 28, 1931, with diagnosis of chronic follicular tonsillitis with glandular involvement, left acute maxillary sinusitis and ethmoiditis. Three days after admission the lids of the right eye were swollen and there was some edema of the conjunctiva. Under local treatment the eye quieted down for three days, flared up again for one day, then stayed quiescent until tonsillectomy and left ethmoid exenteration and intranasal antrotomy, March 9, 1931.

The ethmoids and antrum were filled with pus. Discharged after three weeks' convalescence in hospital. Result: Cured.

Case 2.—D. C. G., Jr., aged 14 years. Past history negative. Present illness: Grippe for two weeks, followed by acute right maxillary sinusitis and ethmoiditis. Admitted to hospital March 30, 1925, with diagnosis of acute right ethmoiditis, maxillary sinusitis with orbital cellulitis and exophthalmos.

X-rays: The plates taken of the sinuses show definite increase in density involving the right antrum and the right ethmoid region. The frontal sinus on this side is larger than the left and there is a slight haziness in its extreme lower portion.

Operations (1): Right antrum opened and washed out, March 30, 1925; (2) Three days later seen by me in consultation and I performed an external ethmoid exenteration and middle turbinectomy. There was an abscess of the ethmoid with partitions completely destroyed. Previous antrotomy opening patent.

Discharged after two weeks. Uneventful recovery. Result: Cured.

Case 3.—I. M. B., aged 14 years. Seen at home February 8, 1929, with acute rhinitis, headache and orbital cellulitis left side. Advised removal to hospital for treatment, which patient's family refused to do. The child grew gradually worse until her condition warranted overruling family's opposition and sending patient to hospital on March 3, 1929.

Diagnosis: Left pansinusitis with orbital abscess and bilateral otitis media.

Operation (March 4, 1929): Incision and drainage of orbital abscess with left external ethmoid exenteration and obliteration of the left frontal sinus with drainage of the left orbital abscess. There was necrosis of the frontal bone and pus in the ethmoids, frontal sinus and upper lid. Cavity packed and wound left open for drainage. Wound closed one week later.

Patient discharged after very stormy recovery of three weeks. Condition improved.

520 COMMONWEALTH AVE.

XCI.

EAR MANIFESTATIONS IN DIABETES.*

V. K. Hart, M. D.,

Charlotte, N. C.

Diabetes will notoriously give eye symptoms, i. e., diabetic cataract and diabetic retinitis. Likewise, it will at times give ear symptoms, viz., vertigo, deafness or both.

Either of these ear symptoms may come from local inner ear degenerative changes incidental to disturbances of metabolism. More commonly, the inner ear changes are secondary to a sclerosis of the nutrient vessels. Sclerosis, of course, is common in diabetes.

We may have these ear symptoms, deafness and vertigo, also, from a true eighth nerve neuritis. This is identical with any other diabetic neuritis, such as sometimes involves one or more motor nerves to an eye.

The differential diagnosis between an inner ear degenerative change (end organ lesion) and a true eighth nerve neuritis can only be made with the galvanic current. We should remember that, as far as we now know, the galvanic test probably acts on both end organ and nerve. It is quite possible then for an end organ lesion to give some diminution of galvanic as well as caloric function. The galvanic test is therefore of most value when absent or nearly absent. In such a case there is probably a true eighth nerve neuritis.

Vertigo may also come from local areas of softening in the cerebellum. We had occasion to observe this postmortem in one diabetic.

However, from a practical standpoint, any inner ear degeneration or eighth nerve neuritis merits investigation from this standpoint, if there is any suggestion in the patient's history of a diabetes. Several times we have been misled temporarily by negative urines, where blood sugar eventually proved to be high.

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*From the Charlotte Eye, Ear and Throat Hospital, Charlotte, N. C.

A typical ear history and examination is summarized.

November 5, 1931. Mr. W. B. M., age 45, white. The patient complains of deafness. He has had noises in the right ear since August and has had some dizziness for two months—not so bad now. He has no colds and very few headaches. The nose, nasopharynx and larynx are negative. There are chronically diseased tonsils with some infection. There are several crowned teeth which should be investigated.

HEARING TESTS

	Right	Left
C	Short 40 seconds	About normal
c4	Short 15 seconds	Short 5 seconds
Bone conduction (Mackenzie Fork)	Short 25-30 seconds	About normal
Audiogram	Loss 31%	Loss 21%
Tubes	Patulous	Same

VESTIBULAR TESTS

Spontaneous	Rotatory, horizontal nystagmus to the left.	
	Right	Left
Minimal caloric	Marked hypofunction	Moderate hypofunction
Galvanic kathodal nystagmus	No reaction up to 10 ma	Increased spontaneous at 4 to 5 ma
Anodal nystagmus	Did not increase spontaneous up to 10 ma	No reaction even at 13 ma

There was, then, nerve involvement of both sides. Wassermann test made by a reliable serologist was negative. (This was confirmed.) Blood pressure, 130/80. This suggests a diabetic neuritis, a focal infection toxemia, a vascular sclerosis or lues of the central nervous system.

Blood sugar, 172 m.g. of sugar per 100 c.c. of blood. Urine, negative.

November 17, 1931. Sugar tolerance test: One-half hour, 333 mgm.; first hour, 400 mgm.; second hour, 217 mgm.; third hour, 204 mgm.

December 29, 1931. Hearing for c4 fork about normal; for Big C fork also much improved. The audiogram shows improvement 13% right and 8% left. Four abscessed teeth were pulled two weeks ago.

The ear examination directed attention to a possible diabetes which was later proven. The improvement under diabetic management is significant. Since four abscessed teeth were extracted, two weeks prior to the last examination, focal infection must also

be considered. However, the improvement was too quick for this, I think, whereas he had been under diabetic treatment by a medical man for nearly two months. Moreover, a focal infection deafness, though not uncommon, does not give, as a rule, such a marked neuritis as shown by great loss of galvanic function. This function may be diminished, but not to such an extent, in focal toxemia.

106 West Seventh St.

XCII.

SAFETY PIN CLOSER AND STAPLE POINT PROJECTOR.*

GEORGE G. CARROLL, M. D.,

ROCHESTER, N. Y.

This is composed of two instruments (Fig. A), one for grasping the safety pin at the spring or hinge, and the other for closing the pin. The same apparatus is designed to grasp the staple at its head, whether it be round or square. The grasping forceps has a pin on each blade that together act as a bumper so that the pin or staple is only grasped at the tip of the forceps and there is no danger of getting too big a bite. This bumper also

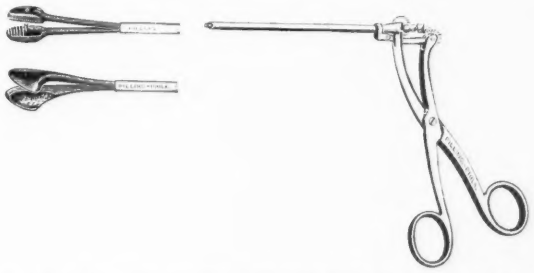

Fig. A. Instrument for grasping safety pin at spring or hinge.

acts as a guide for the forceps to slide down the inside shaft of the staple, whether the staple is parallel to the shaft of the forceps or whether it is tipped to one side. When the forceps are closed these pins insert themselves in corresponding holes in the opposite blade. By using two pins we were able to get a larger opening (1½ cm.) of the blades of the grasping forceps, without any danger of a space in which the foreign body could slip through

*Read before the fourteenth annual meeting of the American Bronchoscopic Society, St. Louis, June 6, 1931.

and become entangled. The second pair of forceps are designed with thin curved blades that are used to grasp the shaft (Fig. B) of the first instrument as a guide to the foreign body. Before pushing the forceps down on the shaft the second forceps are placed on the neck of the set-screw in the handle of the first instrument and covered with the index finger of the left hand (Fig. B). This gives two points of contact and guides the blades directly to the distal end of the first instrument (Fig. C). The blades of the second instrument stop with a click when they reach the end of the shaft of the first instrument. The blades are

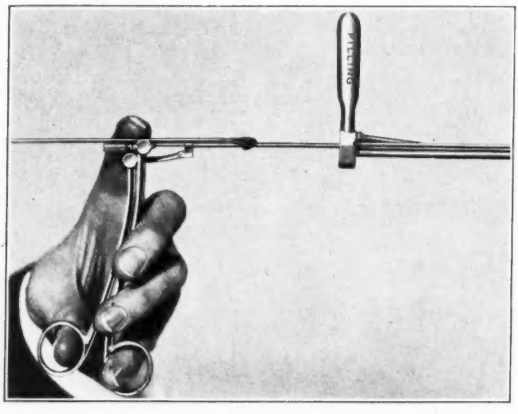

Fig. B. Showing method of adjusting second forceps on the neck of set screw of first instrument.

then slightly opened to release them from the shaft of the first instrument, and by gently pushing forward they ride over the blade of the grasping forceps on two grooves designed for that purpose (Fig. D), and after pushing beyond are opened wide (Fig. E) and drawn back to grasp the pin, which is then closed (Fig. F). The proximal ends of the blades of the second instrument are beveled backwards so that they will not form a hook, whether they are open or shut. The same procedure is followed in covering the roughened ends or points of a staple, except that when the blades of the second forceps, which are serrated on the

inside, are slowly pulled over the roughened ends or points of the staple, a grating sensation is felt in the hands, which helps to control the blades as they cover the roughened ends or points of the staple (Fig. K).

The second instrument is made 7½ cm. longer than the first in order to lock both hands together, so that the slight movement

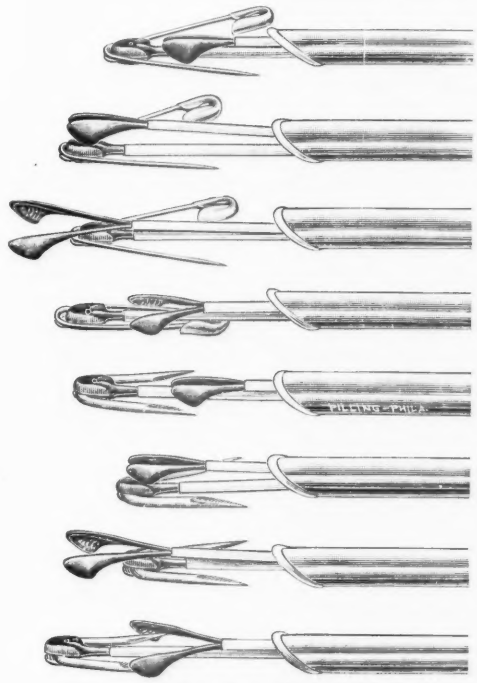

Fig. C, D, E, F, G, H, J, K.
Shows method of adjusting and closing forceps.

of the blades over the roughened ends or points of the staple can be controlled. The fluoroscopic orientation should not be overlooked.

The principle involved in using the shaft of the first instrument as a guide to the second is new, as far as I can determine, and

helps the operator to immediately orientate himself as to the position of the grasping forceps in relation to the foreign body without seeing it.

In conclusion, would say that the instruments should be thoroughly inspected before attempting to use them on foreign bodies. The shafts should be perfectly straight and not bent or curved, and the shaft of the first instrument should be thoroughly cleansed and free from any tarnish, so that the blades of the second instrument will slide down to the foreign body with the least amount of friction. While using do not bend the shafts of either instrument—keep them parallel and then they will automatically work as described.

I am indebted to Mr. Charles J. Pilling of the George P. Pilling & Son Co., Philadelphia, for valuable assistance rendered to me in the construction of this instrument.

XCIII.

The Cause of Otosclerosis.*

(Continued.)

Louis K. Guggenheim, M. D.,

St. Louis.

Hagens[1] reports certain interesting staining reactions in otosclerotic foci. He states that his researches could not reveal with certainty the presence or absence of calcium, as such, in otosclerotic foci. The tests with alizarin and purpurin gave negative reactions. The ammonium sulphide test and the prussian blue reaction proved definitely the presence of iron in the bone; therefore the blue reaction in the foci stained with hematoxylin-eosin may be due to iron. "The test in which mordanting with alum was followed by staining with hematein indicated that the affinity of the focus for the colloidal aluminum hydroxide caused the adsorption of the hematoxylin." This may cause the focus to take a deep blue stain and may explain the blue reaction in the absence of iron. Either the presence of iron in the focus or the adsorption of hematoxylin through the medium of the colloidal aluminum hydroxide would account for the peculiar blue reaction with hematoxylin-eosin. The foci seem to have an increased power of adsorption for the iron and for aluminum hydroxide not possessed by normal capsular bone. The presence of iron in the foci may be due to freed blood left in the areas through the marked vascularity, or iron freed from tissue cells during the destructive process. Needling as a decalcification test may explain the presence of iron or the acid used for decalcification may liberate iron from the tissues.

Hans Brunner,[2] in an article entitled "Ueber Spaltbildung in der knöchernen Innenohrkapsel," quotes O. Mayer as having pointed out spontaneous fractures in the following locations: (a) in the region of the posterior ampulla; (b) in the promontory

*From the Oscar Johnson Institute of Washington University.

from the niche of the oval window to the round window; (c) above the oval window; (d) numerous ones in the canal region (superior and posterior canals); (e) in the bony spiral plates of the cochlea and in the internal auditory meatus.

In order to substantiate the findings of Mayer, Brunner examined his own collection of twenty-six serially sectioned temporal bones. The youngest was 9 years, the oldest 71 years. Fractures of the promontory were found in only two bones. Three bones showed an oblique fissure from canalis facialis to the superior ampulla. Fissures found in Paget's disease are not considered.

Fissures in the canal region were seen in twenty-four bones (majority of bones). As a rule the fractures pass from endost outward. The fissures often show a lumen (artefact). In eleven bones fissures were found in the region of the round window. All the fissures in the round window region have this in common: they lie in the median wall of the window niche in the subiculum promontorii.

Brunner explains the fissures as follows: (1) They may occur around vessels or bundles of connective tissue in the bone. (2) They may occur in the sense of O. Mayer (spontaneous fractures). Brunner doubts that the fissures are really spontaneous fractures due to some mechanical disturbance of the temporal bone: no blood, pigment or any reaction processes are noted. Brunner thinks the fissures are either canals for vessels or are dehiscences. He cannot agree that a callous formation from these fissures is related to otosclerosis: first, because the fissures are most common in the canal region, where otosclerosis is more seldom seen, and rarely in the oval window region, where otosclerosis is most common. Again, the most common site of traumatic fissure is the tegmen tympani, where otosclerosis has not been found.

F. Leiri[3] tells us, in his "Über Änlichkeiten in den Krankheitsbildern der Otosklerose und der juvenilen Epiphysen-affektionen," that the labyrinth capsule, in view of its small dimensions, is exposed to a marked mechanical strain. This is chiefly from the permanent tension of the strands of the membrana basilaris in the basal turn and through the ever present tug from the movements of the stapes and the membrane of the round window (con-

tinuous noises in our environment). The occurrence of otosclerosis in the region of the basal turn, oval and round windows is the result of this chronic insult. In an earlier work the author mentioned the similarity betwen otosclerosis and juvenile epiphysis disease (osteochondritis deformans juvenilis). Characteristic for juvenile osteochondritis is that the involvement begins in the regions where growth of the bone occurs, namely, in the region of the epiphysis line or in the joint cartilage. He then asks:

1. Do there exist in the labyrinth capsule growth zones analogous to the epiphysis line and joint cartilage?

2. Do otosclerotic foci arise in these growth zones?

To both questions he answers yes. At birth the cartilage-preformed labyrinth capsule is complete as to size and form; at this time there still exist cartilage rests in the enchondral capsule. By the second year ossification is complete. The growth zones in the capsule comparable to the epiphysis line and joint cartilages of the extremities of the juvenile individual, must therefore be searched for in the embryo (growth period of the labyrinth). During the fifteenth, sixteenth and seventeenth week of embryonal life, when ossification is well under way, there occur cartilage areas similar to the epiphysis line and joint cartilage. These are the cartilage partitions in the embryo capsule, whose topography and histology are known through the work of Mayer, Wittmaack, Kosokabe and Eckert-Möbius. Through studies of the cartilage partitions of the capsule the writer has come to the conclusion that they form two more or less protruding cartilaginous parts which in sagittal direction are on both sides of the vestibule, and at the beginning of the basal turn, passing between the region of the window and the carotid knee.

Of these partitions the superior and lateral (the Mayer partition) lies in the labyrinth wall between the tympanum and vestibule. It is most strongly developed in front of the oval window and reaches to the window edge. At this point it is quite wide and reaches upward almost to the dura of the middle fossa and downward medially as far as the beginning of the scala vestibuli. It also reaches far forward between the middle and basal turns to the anterior portion of the cochlea and posteriorly past the

oval window, medially to the facial canal and as far as the semicircular canals. The inferior medial partition (the cartilage rest in front of the synarthrosis petrobasalis), according to Eckert-Möbius, begins median to the anterior inferior part of the cochlea near the carotid knee and passes backward median to the inferior part of the median wall of the basal turn. These partitions pass posteriorly under the internal auditory meatus to the region immediately median to the round window. Also posterior to the round window is a piece of the partition which reaches as far as the region of the posterior canal ampulla. These cartilage areas the writer believes are comparable to the epiphysis and joint cartilage and are growth zones.

Leiri then calls attention to the fact that the foci which, according to Mayer and Kosokabe are the otosclerotic areas, are all in the cartilage growth areas; the otosclerotic foci therefore develop in relation to the growth zones, just as do the foci in juvenile osteochondritis.

The most common area for otosclerosis is in front of the oval window, where is located the most extensive and often postembryonally persisting cartilage partition. The writer admits that histologically the juvenile epiphysis affection and otosclerotic bone are not exactly the same, but the same elements may be found: connective tissue, osteoclasts, osteoblasts and new formed basket-weave bone. Both processes begin in incomplete bone (bone with cartilage still present). In summarizing, the writer states that although he believes that mechanical irritation plays an important rôle in the development of otosclerosis it is certainly not the basic cause.

J. K. M. Dickie,[4] in a summary of bibliographic material available for 1930, in the field of progressive deafness, etc., quotes Lange[5] as follows: In a study of the early changes in otosclerosis (material consisting of sixty-six temporal bones from forty-five patients) Lange found the site of foci as follows: Oval window alone, twenty-two; both windows, seven; round window alone, four. In only two of the forty-five cases were the windows free. In nineteen of the forty-five cases blue-staining bone was found in the canals; of these, five were typical foci while fourteen were

in the first stage. Of twenty-one cases, in which both sides were examined, twenty showed symmetrical areas of involvement. Lange studied the areas away from the promontory for early changes. The promontorial changes showed more hypertrophy. Here the enchondral bone is very thin and receives a better supply of blood from the surface vessels. Hypertrophy elsewhere is limited by the other surrounding bone. Irregularity of the new bone corresponds to branches of vessels. Giant cells may be found but not commonly. Normally in the enchondral areas bone cells are scarce by comparison with ordinary skeletal bone and the long processes are not well seen. Otosclerotic areas are rich in cells.

THE RELATION OF OTOSCLEROSIS TO GENERALIZED BONE PATHOLOGIES.

Otosclerosis is a bone condition involving first resorption with subsequent formation of a different type of osseous tissue. Although the capsule of the labyrinth differs from other bones in the body in that it is more compact and harder than bone elsewhere and because of its inclusion of lamellarless, fine-fibered strand bone and cartilage rests throughout life, it may be pathologically involved in a manner quite identical with osseous involvement elsewhere. In osteodystrophia fibrosa (von Recklinghausen's disease and Paget's disease) the same resorption and filling in with fiber-bone occurs as in otosclerosis. In osteogenesis imperfecta congenita we have a maldevelopment (fiber-bone in place of lamellar bone), as in otosclerosis, etc.: therefore, one can hardly conceive of a more logical, scientific attack upon the problem of otosclerosis than a careful analysis of the changes occurring in generalized dystrophies of the osseous system and a comparison of these changes with the changes observed in otosclerosis.

Moritz Weber has concerned himself so profoundly with these comparisons that the reader will be familiarized best with this phase of the subject by quotations from the publications of this writer.

Weber[6] states, "Research into the etiology of otosclerosis requires a knowledge of the final construction and development of the bony capsule." Then follows:

(A) GENERAL PRINCIPLES OF NORMAL BONE FORMATION.

1. Undifferentiated but potential mesenchyme cells. These are spindle cells with clear nuclei lying in an intercellular substance.

2. Differentiation of mesenchyme. Between the mesenchymal cells intercellular substance increases. There are two types of intercellular substances: one stains red with eosin, the other blue with alum-hematoxylin.

3. The formation of "nuclei." The appearance of two different intercellular substances represents the first step in the development of centers for subsequent formation of bone. The second stage is the calcification of matrix. Weber has termed the two centers of bone formation "nuclei." One (eosin-red matrix) is the connective tissue (fiber-bone) nucleus; the other (alum-hematoxylin-blue matrix) is the cartilage nucleus.

Fiber-bone "nucleus" develops as follows: the cells embedded in the eosin-red matrix take on a jagged appearance; the intercellular substance shows an increased density and stains more deeply. Finally the cells are connected by small anastamoses and the intercellular substance diffuses to the undifferentiated cells. The fibrils of the surrounding connective tissue penetrate into the enlarged and compact basic matrix and form there a dense irregular mesh. As soon as the intercellular substance calcifies the connective tissue "nucleus" is completed. It is then called calcified connective tissue (Schaffer), rootstock (Gegenbauer) or fiber-bone (Weidenreich). The original periosteal bone is fiber-bone. Later through rows of osteoblasts lamellar bone is formed.

The Cartilage "Nucleus."—Differentiated mesenchyme cells embedded in blue matrix are the starting point of the cartilaginous "nucleus," which develops as follows: The embedded cells enlarge and assume a vesicular structure. Several nuclei may be noted in one cell.

The Enchondral "Nucleus."—The primary enchondral "nucleus" forms when the cartilaginous model is opened up by invasion of blood vessels. The cells enlarge and appear vesicular. The intercellular substance increases, becomes dense and stains more deeply. Blood vessels enter the cartilage model and take with them undifferentiated but potential connective tissue cells (periosteal

bud). The whole cartilage model is hollowed out and the primary marrow space is the end-result. The intercellular substance of the cartilage then calcifies and represents the primary enchondral "nucleus."

Lamellar bone is laid down on the above "nucleus." There is only one mode of lamellar bone formation and that is through osteoblasts, arranged in epithelioid rows. At first the bone substance is soft (osteoid) but later it becomes calcified. In the completed bone the osteocytes are composed of former osteoblasts. Weber classifies bone into (a) lamellar fiber-bone (parallel fibers of periosteal bone), and (b) shell-bone of Weidenreich. (In later papers he has recognized Max Meyer's description of capsular construction.)

The lamellar fiber-bone is typical periosteal bone and is characterized by numerous connective tissue fibers (Sharpey fibers) and vessels which are embedded as canals of Volkmann.

Resorption of Bone.—The lamellar fiber-bone is slowly resorbed from the inside, by osteoclasts, which probably attack by means of ferments and form Howships' lacunæ. Vessels may also help in the erosion.

Shell-bone Formation.—Shell-bone is lamellar bone, formed by osteoblasts around blood vessels, resulting in the Haversian systems. Since the normal marrow is not fibrous, the lamellæ of the shell-bone are free from connective tissue fibers.

Transformation of Shell-bone.—In the course of skeletal development the early formed osteons (Haversian units) are slowly resorbed. The spaces are filled with new shell-bone. In advanced age the lumens of the osteons are narrowed and may finally be obliterated.

The End-result of Bone Development.—A completed bone reveals a complicated histologic picture which cannot be appreciated if one does not thoroughly understand the various phases of its development. For illustration, in a transverse section of a human long bone of an infant, an overlapping of all the stages mentioned above, is present. The outer or subperiosteal parts are lamellar fiber-bone (except for fiber-bone where tendons are inserted). The lamellar fiber-bone is replaced from within by shell-bone but fragments remain. Toward the marrow, one may

observe a conglomerate mass of osteons, whole or fragmentary, and the remains of perichondral "nucleus." Close to the marrow are the osteon-lamellæ of the marrow, which continue into the spongiosa.

General Principles of Abnormal Bone Formation.—In general, under pathologic conditions, bone formation and resorption exceed the normal rate, but in principle the process is a repetition of the normal. Hereditary factors may influence the germinal tissue, leading to disturbances in the undifferentiated but potential mesenchyme cells. These cells may lack the ability to form the supporting system required for proper bone development or they may form bone where it does not normally occur. An abnormal appearance and increase in intercellular substances is frequently observed. There may be a deposition of soapy material ("Fettkalk," Aschoff). Such deposits may follow inflammation, necrosis and disturbance of metabolism.

There may be a complete absence of lime deposits in cartilage "nuclei" (etiology unknown), which is seen in chondrodystrophia foetalis. An incomplete calcification in the preparatory zone is observed in rickets. Deficient calcification may result from endocrine disturbances, as in nanosomia pituitaria and nanosomia athyreotica. A rapid and increased calcification has been found in gigantism.

Pathologic Appearance of "Nucleus" Formation.—According to Borst, metaplasia represents the development of a fully established tissue into one of a different character. In a general way metaplasia is a throwback to ontogenetic and phylogenetic phases of development. The metaplasia of connective tissue into bone substance is of the greatest significance in the study of otosclerosis. The metaplasia of cartilage to bone has not yet been proven.

Wherever in the body, intercellular substances such as hyalin, colloid, collagen, etc., make their appearance, it is a result of a pathologic process. "Nuclei" formation may take place, provided the substance becomes calcified. Any tissue may thus develop into a center for subsequent bone formation.

Disturbances of the Lamellar Fiber-bone.—The most frequent disturbance of the periosteal bone is an intensified ossification

which increases the diameter of the corticalis. The newly formed bone is in its first stage a fiber-bone.

A reduction of periosteal ossification may decrease the corticalis. It is found in osteogenesis imperfecta, in the two vitamin deficiencies, Möller-Barlow and scurvy.

Disturbances of the Shell-bone.—In cases where bone development remains at the stage of the cartilage model and shell-bone is diminished or absent, dwarfism may result. It has been observed in cretinism. An important disturbance of shell-bone formation is its deficiency in calcium, which leads to the apposition of osteoid. Osteoid is always found in natural and experimental rickets and in osteomalacia and may be present or absent in osteodystrophia fibrosa.

Pathology of Resorption.—Increased resorption by osteoclasts is one of the most frequent disturbances of resorption. It is common in inflammation of bone, in tumor invasion, in pressure atrophy, etc.

New types of resorption: (1) Vessels have the power of eroding bone; (2) halisteresis—a diffuse decalcification called by Recklinghausen, trypsis. Some doubt that it exists. It is supposedly due to the action of a ferment.

Weber[7] reports a case of osteodystrophia fibrosa showing pseudotumors and cyst formation. The condition was of the hyperostotic-porotic form and occurred in an 8½ months old Boston terrier. There existed a progressive bone atrophy (picture of dissection atrophy of L. Pick). Weber outlines the various stages of bone development, beginning with undifferentiated mesenchyme, (1) differentiation, (2) "nucleus" formation, (3) shell-bone formation, (4) resorption. The "nuclei" comprise calcified cartilage and calcified connective tissue. Resorption is positive functional accomplishment of the bone building apparatus. The stage of differentiation of mesenchyme showed marked prominence (fibrous tissue). Also there was a great increase in undifferentiated mesenchyme.

Weber looks upon osteodystrophia fibrosa as an irritation and a left deviation of the bone picture. To an unknown irritation (disturbance of metabolism) the bone building system answers with resorption. To cover the defect all the reserve is mobilized

down to mesenchyme. There follows formation of fibrous tissue, then fiber-bone. Shell-bone does not form again; at any rate, not further than osteoid. The mesenchymal sponge of the pseudotumor is the highest expression of "Linksverschiebung."

The underlying factor for the development of this generalized disease must be looked for in the power of differentiation possessed by the mesenchymal tissue. The focus of origin may be in the periost or endost. The culmination point is reached when the shell-bone of the old cortex has completely disappeared and the substitution with fiber-bone has been completed. Healing consists of discontinuance of resorption and a relative "Rechtsverschiebung" of the bone picture (reappearance of shell-bone osteoid).

Osteodystrophia fibrosa is supposed to develop upon the basis of a chronic local or generalized disturbance of metabolism. The disease should be considered analogous to the kidney disease with osteopathia fibrosa.

To solve the question of etiology it would seem most rational to try dietary experiments. The value of diet is, however, questionable because of the fact that metabolism stands between a possible endogenous causa and the effect upon the osseous system. The dietary experiment should consist of an endeavor to find which foods are capable of causing an irritation and "Linksverschiebung" of the bone-picture.

Weber's statement that the underlying factor in the development of osteodystrophia fibrosa must be looked for in the power of differentiation possessed by the mesenchymal tissue, assumes tremendous importance in the present research. In osteodystrophia fibrosa something causes resorption of normally developed bone, and there follows a filling in with an inferior bone (fiber-bone) even to excess (hyperostotic form—von Recklinghausen's disease). The picture of osteodystrophia fibrosa is, according to Weber, identical with otosclerosis in which there has also occurred resorption and filling in with an inferior bone. In osteodystrophia fibrosa the cause of resorption, Weber believes, is some disturbance of metabolism, the filling in occurring through mesenchymal activity. The present research is striving to prove that in otosclerosis not only does the filling in result from activity of the

pluripotential mesenchyme cell but the resorption itself is the result of a regressive characteristic of the mesenchyme cells in certain individuals, resulting in secondary activity of those elements.

Weber,[8] in his article concerning the relationship of otosclerosis to osteodystrophia fibrosa, quotes O. Mayer as stating, in 1928, that otosclerosis has a connection with osteitis deformans of Paget and that where both conditions were found in the same patient it may have been a coincidence but more likely a genetic relation existed.

Although osteodystrophia fibrosa is usually a generalized disease, it has been shown by Frangenheim, Kaufman et al. that a localized form exists which may be balanced against otosclerosis. Weber believes that otosclerosis is primarily a problem of bone pathology rather than otology. His paper deals with the temporal bone changes in a dog with generalized osteodystrophia fibrosa (previously described). The same changes were observed in both temporal bones. Ossicles and drum membranes were normal. There was no ankylosis of stapes, no calcification of the annular ligament. The tympanic mucosa was not thickened. Tendinous insertions showed resorption by giant cells and fibrous marrow with hyaline islands was found. Such foci originated in the periosteum and endosteum. The bone bounding the capsule contained marrow, chiefly of lymphoid and fat tissue. However, where the marrow lay next to the fibrocartilaginous partitions it became fibrous. The spaces of the spongy bone were filled with normal bone marrow. One portion showed dissecting atrophy. The bone surrounding the labyrinthine capsule showed, as did the other bones of the skeleton, a typical osteodystrophia fibrosa. The capsule showed a rarefaction. Large and small irregular cavities with evident relationship to the vascular system were found. Within the cavities were giant cells resorbing bone, hyperemic vessels, embryonic connective-tissue or fibrous marrow. Here and there a small osteoid margin bounded the resorption cavity. The osteoid was separated from the true bone by a line of cementum. A large cartilaginous remnant was found on both sides, beneath the horizontal canal. About in the center of the semicircle were several cystic spaces surrounded by hemorrhages and infiltration with round cells. Toward the loop was connective tissue contain-

ing few nuclei and much hyaline intercellular substance. The hyaline connective tissue abutted on the cartilaginous remnant. The cartilage cells were vesicularly distended. The intercellular substance was calcified and stained deep blue with hematoxylin. Toward the connective tissue it became granular. Next to the cartilaginous focus was a large resorption cavity lined with osteoid deposited in lacunæ. The osteoid was lined with osteoblasts toward the lumen. The cavity was filled with vessels, loose young connective tissue and fibrous marrow. Preliminary resorption had ceased and formation of new bone instituted. The deposit of lamellar bone was minimal. From the normal marrow directly in front of the cochlea a vessel was noted leading from old normal marrow into a resorption cavity. Lining the walls of the canal was blue bone, with no cementum line, and toward the vessel this gradually changed to pink osteoid. The resorption cavity was filled with new shell-bone separated by cementum from the old bone. The lumen was lined by a narrow ribbon of osteoid. The wall of another perforating canal showed a granular crumbly zone staining dark blue. This gradually merged into old bone without cementum.

The bone cells here showed no halo of protoplasm, which seemed to have merged with the osteoid matrix. Within the resorption cavity was found a typical osteoplastic marrow and numerous osteoblasts.

Theoretically two processes might be assumed to explain the origin of the blue and pink zones: (1) degeneration of mature bone normally developed from mesenchyme, and (2) new formation of immature bone maldeveloped from mesenchyme. Two processes interrupted by death are apparent decalcification and calcification. Weber likes to regard the areas, not separated from the surrounding bone by cementum, as degenerated bone. Where a cementum line is present, one is probably dealing with a new formation of maldeveloped bone.

Where the intercellular substance of new bone is not the same as the cementum line, the latter is visible; where it is the same, the cementum line will not be visible. The direction of fibers under polarized lite will determine whether the bone is new or old. In the present case the capsule showed more resorption than

new bone formation (hypo-ostotic porotic form—Christeller). Where new bone formation exceeds the resorption it is called hyper-ostotic porotic.

Comparison of osteodystrophia fibrosa and otosclerosis: In osteodystrophia fibrosa the disease results from a chronic local or general dystrophy. The vascular system acts as the mediator between dystrophy and the degeneration. Especially has venous stasis been held as a cause (von Recklinghausen, Pommer, Lang et al.).

In otosclerosis several authors believe a softening of the bone initiates the process. The theory of venous stasis is championed by Eckert-Möbius, Gibelli, Grünberg, Heyninx, Lange, O. Mayer (1911), Wittmaack et al. Some believe an enlargement of the haversian canals the initial stage. Weber agrees with Döderlein that the blue coats around the vessels are probably degenerated bone. In Weber's present case were observed blue areas along the vessels, which may be identified with the "granular crumbly zone" of Pommer, and which must be interpreted as evidence of degeneration.

The Reaction.—Resorption by osteoclasts is an important step in the normal development of bone. In osteodystrophia fibrosa an unknown cause leads to a stimulation of the functional bone-building system which finds its expression in the increased appearance of giant cells. They produce the spaces in which the new bone forms. This increased lacunar resorption is never found in rickets or osteomalacia. Weber agrees with Mayer that these two conditions show no relation to otosclerosis.

Recently the view has predominated that before the appearance of osteoclasts there must occur a "readiness" (degeneration) in the bone. This produces the stimulation which leads to the appearance of osteoclasts. Possibly blood vessels produce the degeneration and the osteoclasts remove the débris.

Appearance of Mesenchymal Tissue.—In both osteodystrophia fibrosa and otosclerosis the cavities produced by osteoclasts and blood vessels are filled with mesenchymal tissue. Teleologically the bone-forming system mobilizes its reserve forces as far back as the youngest stage in order to repair the defect. The mesenchymal tissue and giant cells arise simultaneously.

The Reparation: Progressive Stage (Differentiation).—At first fibrous marrow develops from mesenchyme. The elements provided for the formation of the vascular system take part in the differentiation. This process is referable to the pluripotentiality of mesenchymal tissue (Hueck, Herzog). The histologic picture resembles granulation tissue both in osteodystrophia fibrosa and otosclerosis. The process, however, is not inflammatory but dystrophic formation of "nucleus." In normal bone development an increase and differentiation of intercellular substance appears with differentiation of mesenchymal cells. Weber thinks this increased intercellular substance is rather a product of a humoral or colloid chemical process than the activity of already differentiated cells. Two products result which when calcified represent (1) calcified connective tissue or fiber-bone; (2) calcified cartilage.

In the resorption cavities of the petrous bone, hyaline islands were observed which Weber calls fibroblastic osteoid islands.

Regressive Phase (Disturbance of Differentiation).—If dystrophy is the cause of these diseases it continues throughout the process and disturbs especially the newly arisen differentiation. On the other hand, the theory has been propounded that the regressive changes may be traced back to a primary hereditary faulty mesenchyme (Mayer). In this case the mesenchyme would lack the ability to form the highest type of bone. As proof of this, blue sclerotics and osteopsathyrosis have been offered. Since these do not occur constantly in otosclerosis the question is not decided.

Regression comprises (1) the lack of differentiation, (2) faulty differentiation.

(1) The normal osseous development reaches its climax with the development of lamellar bone (shell bone—Weidenreich). This occurs from osteoblastic activity (epithelioid arrangement). The highest unit is the haversian system (osteon). In osteodystrophia fibrosa the osteon did not appear at all or as only osteoid. The same has been observed in otosclerosis. In osteodystrophia fibrosa, as well as in otosclerosis, one observes a lack of differentiation by the absence of or defective formation of lamellar bone.

(2) The mesenchyme, filling the resorption cavities and closely related to the vascular system, is pluripotential. It differentiates

in osteodystrophia fibrosa in a progressive way up to different levels; then the progressive tendency exhausts itself and gives way to regression (maldifferentiation ensues). The product of this faulty differentiation is hamartoplasia. As hyperplasias they represent intermediate formations between true tumors and normal tissue on the one hand and between inflammatory repair tissue and normal tissue on the other. The dense fibroma-like areas within the epulis and the blue islands of granular, crumbly bone represent such malformation.

Now, concerning otosclerosis, are there also regressive changes? Mayer stated that otosclerotic foci originate through the pathologic growth which has been observed in connection with embryonic malformation.

Are there histological proofs of this theoretical assumption? Otosclerotic foci present histologic differences. These differences may be explained by assuming that the progressive phase is interrupted by regression at various stages. When large areas of fibrous marrow, poor in cells, are found they may be compared to the fibroma-like areas of osteodystrophia fibrosa. In the sclerotic form there is newly formed bone. The marrow spaces contain few cells except for osteoblasts. The matrix of this new bone, staining deep blue with hematoxylin, has been described as reticulated. Its cells are large and irregular. This bone may be identified with the islands of granular, crumbly concrement bone and the malformed fiber-bone, as observed in osteodystrophia fibrosa.

In osteodystrophia fibrosa and in otosclerosis "nucleus" formation occurs, during which intercellular substance as well as cells undergo regressive changes. The new-formed bone in otosclerosis stains blue (H). Mayer believed this is due to an abundance of cementum. In the "nucleus" formation no real braided fiber-bone is found but rather a malformation which might be called a cemented connective tissue. Mayer observed a cystic form of otosclerosis, as well as a vascular form, reminding one of the appearance of cysts and blood lakes in the mesenchymal sponge in cases of osteodystrophia fibrosa. "The entire histogenesis in the one is identical with that in the other. Otosclerosis is to be balanced against a local form of osteodystrophia fibrosa."

Weber defines hamartoplasia as a malformation from newly formed mesenchymal tissue; a congenital maldifferentiation is hamartie; congenital maldifferentiation with hyperplasia is hamartoma; congenital maldifferentiation with tumor formation is hamartoblastoma. Hamartoplasia is then a pseudotumor of bone which has developed by maldifferentiation from a mesenchymal tissue which is not a congenital remnant but a new appearance during life, and results from irritation of the bone-forming system. "The end-picture of otosclerosis should be regarded as the picture of hamartoplasia."

Mayer championed this theory in 1911, but later abandoned it in favor of his hamartoma theory.

In regard to changes, particularly around vessels, Weber states one must distinguish between two pictures of different origin: (1) old, degenerated bone, and (2) new maldeveloped bone. According to Siegmund, the symptom complex of osteodystrophia fibrosa develops whenever, under certain circumstances, bone comes to be resorbed. Blood vessels may act as mediators, leading to a degeneration. There is a close relationship between the development of otosclerotic foci and cartilaginous remnants, which suggests that possibly, in contrast to bone, cartilage remnants are more sensitive to dystrophic influences. This plus the peculiar distribution of blood vessels may explain the selective involvement of the labyrinthine capsule and therefore the focal nature of otosclerosis.

Weber goes into a most interesting and complex discussion of the comparison of von Recklinghausen's disease, in which resorption is followed by new bone formation and pseudotumor, Paget's disease with resorption and new bone formation but no pseudotumors, and otosclerosis which is either a hyperostotic porotic local manifestation of a generalized von Recklinghausen's disease or, if not hyperostotic, it is no longer to be considered as otosclerosis superimposed upon a von Recklinghausen's disease but as a simple part of the latter. Where in such a case we have ankylosis of the stapes we are dealing with clinical otosclerosis, but histopathologically no otosclerosis but a part picture of a generalized osteodystrophia fibrosa.

Finally it may be repeated that Weber holds strongly to a theory that underlying otosclerosis and osteodystrophia fibrosa there is a common cause and an identical histopathology; that Paget's disease, von Recklinghausen's disease and otosclerosis are closely related; that in Paget's disease a resorption with new bone formation may occur in the otic capsule just as elsewhere in the body; that pseudotumor formation must occur for the disease to be called von Recklinghausen's; that in a generalized osteodystrophia fibrosa the otic capsule may be involved with even ankylosis of the stapes, but if no pseudotumor formation occurs, the localized involvement cannot be called otosclerosis.

Weber,[9] in his "Theory of Experimental Otosclerosis," emphasizes, in the summary, that—

1. The foundation for judgment as to bone changes rests upon a knowedge of the normal bone picture. This comprises certain time phases of the bone-building system. Bone changes may be judged from the standpoint of time, location, quality and quantity. The hypothetical synthesis of these factors gives the pathologic bone-picture.

2. The bone picture of otosclerosis is postfetal or fetal; local (on typical areas within the labyrinth capsule); shows the result of irritation and maldifferentiation of the bone-building system with relative or more frequently absolute "Linksverschiebung" of the bone picture (fiber-bone formation).

3. The bone picture of generalized osteodystrophia fibrosa (so-called ostitis fibrosa) is postfetal, generalized irritation and maldifferentiation of the bone-building system with relative or absoute "Linksverschiebung" of the bone picture. The exciting cause lies, supposedly, in a generalized disturbance of metabolism. In cases of hyperostotic osteodystrophia fibrosa the "Linksverschiebung" is absolute.

4. The bone picture of local osteodystrophia fibrosa is the same as the generalized form and results from local irritation. Presumably the local form is due to some local disturbance of metabolism.

5. The bone picture of local osteodystrophia fibrosa may appear in a nonhyperplastic or a hyperplastic form. The latter occurs as "brown tumors." These are pseudotumors.

6. The bone picture of otosclerosis is identical with local osteodystrophia fibrosa. Otosclerosis may be spoken of as osteodystrophia otosclerotica. In a few cases it appears as the nonhyperplastic form of osteodystrophia fibrosa, but in the majority of cases it presents the hyperostotic-porotic form.

7. The occurrence of a pseudotumor in a case of generalized osteodystrophia fibrosa is the result of a local increase in the systemic dystrophic disturbance of metabolism (through degeneration products in cartilage?).

8. The bone picture of osteogenesis imperfecta is fetal, generalized inhibition and maldifferentiation of the bone building system with relative "Linksverschiebung" of the bone picture. The difference between the so-called congenital form and the late form (osteogenesis imperfecta tarda, idiopathic osteopsathyrosis, brittle-bones) is only a matter of degree. The late form is the milder form. Both conditions are fetally predetermined.

9. Comparing the bone picture of osteogenesis imperfecta with that of osteodystrophia fibrosa, it appears that there is similarity in respect to maldifferentiation and relative "Linksverschiebung," but in the former there is inhibition; in the latter an irritation of the bone-forming system. In order then for a local osteodystrophia fibrosa to occur in a case of osteogenesis imperfecta there must occur some local irritation.

10. At the site of fracture (mechanical irritation) in osteogenesis imperfecta there develops the picture of local osteodystrophia fibrosa. Through increase of the local irritation there may even occur the picture of the local hyperplastic form (pseudotumor).

11. The final picture of otosclerosis may result in various ways. It is therefore first necessary to find an experimental procedure which will lead to the final picture of otosclerosis and by further experimentation to find a common etiologic factor in all the conditions above discussed.

12. The synthesis of the bone picture of otosclerosis will most probably be accomplished if the following three postulates could be complied with:

(a) There must be a sufficient amount of cartilage rests in the experimental animal's ear capsule.

(b) A generalized disturbance of metabolism in the sense of an osteogenesis imperfecta must be brought about.

(c) There must be accomplished an early enough chronic local irritation (disturbance of metabolism) in the region of the cartilage rests.

13. Concerning the possibility of accomplishing this—

(a) Either very young animals must be used or a special type of animal must be found, which in its early life shows an extensive cartilage rest formation. If this is not accomplished, then the disappearance of cartilage must be artificially prevented; perhaps through disturbance of internal secretion (thyroid extirpation?).

(b) Inasmuch as the bone picture of osteogenesis imperfecta resembles very markedly fetal scurvy, perhaps newborn animals with fetal scurvy could be used.

(c) The local irritation will probably be best accomplished through stasis (operation, Wittmaack's).

14. A synthesis may be expected without operation or special irritation if in the presence of sufficient cartilage rests a disturbance of metabolism, in the sense of a generalized osteodystrophia fibrosa, could be brought about.

15. It will probably be impossible to produce otosclerosis in the presence of normal general metabolism. It is questionable if otosclerosis can exist in a case with normal general metabolism.

Moritz Weber,[10] in his work entitled "Otosklerosis in polarisiertem Lichte," seems to have settled the question, once for all, that otosclerotic bone is a new-bone formation, sharply delimited from the original capsular bone. In his summary he states that the use of hematoxylin-eosin staining does not give full bone details and that polarized light, used on decalcified sections, gives us a true picture of the fibrils of the capsular bone. In ten cases he used the combination of the two methods and discovered a series of new facts. Instead of lamellar bone heretofore described, he found a fine-fibered, lamellarless, so-called strand-like marrow bone, first described by Max Meyer. The new name of strandshell bone is suggested. Where, in hematoxylin-eosin stains, the blue-stained bulge of otosclerotic bone with no cement line meets the normal bone one notes with polarized light that the

fibers of normal bone stop short as if cut off. The normal bone shows cavities into which the otosclerotic bone fits. The otosclerotic bone is composed chiefly of a framework of large bundles of fiber-bone. Lining the cavities are a fine-fibered shell bone partly without lamellæ (strand-bone). Otosclerosis is a new growth following a previous resorption. Cement lines are absent in most parts. This does not speak against the fact that otosclerosis is a new growth, for the sharply ending fibers in polarized light prove the contrary.

In polarized light osteodystrophia fibrosa presents the same picture. In specimens stained with hematoxylin-eosin, osteodystrophia fibrosa presents, however, numerous cement lines (mosaic structure). Weber feels there is in otosclerosis a disturbance of cement formation. The blue coats of otosclerosis represent new-growth following resorption of capsular bone.

CRITIQUE.

Anyone who has carefully studied Weber's numerous publications on bone pathology cannot fail to be greatly impressed by his carefully prepared articles. Few men have his ability to transmit to others the details of such a complex subject as bone disease. Although the writer has not had the opportunity of studying Weber's specimens, his well-made photomicrographs speak forcibly for the identity of the bone picture of osteodystrophia fibrosa and otosclerosis and disprove the contention of Wittmaack that no light can be thrown on the problem of otosclerosis by the study of bone processes elsewhere in the body.

Weber states that because osteodystrophia fibrosa and otosclerosis show identical histologic pictures is by no means proof that there is a common etiology. In the summary just given, however, he makes it plain that he believes otosclerosis due to a disturbed metabolism and that it can well be that underlying osteodystrophia fibrosa and otosclerosis there is a common cause.

When Weber states that it is questionable if otosclerosis can exist in a case with normal general metabolism, one's mind drifts back over a great many years of clinical experience and concludes, perhaps unscientifically, that most of the typical otosclerotics were pretty healthy people except for their ears; however, a metabolic

disturbance could have existed in early life when the process was initiated. Further, otosclerotics have not shown any bone pathology elsewhere in the body, which should certainly be present if otosclerosis is due to a generalized disturbance of metabolism affecting bone. It is rather difficult to conceive of a local, bilaterally identical pathology due solely to a disturbance of metabolism, and we do know that in most cases the otosclerotic involvement is bilateral and identical.

Weber states that otosclerosis is either fetal or postfetal, but does not include the condition in the category of hamartomas (hyperplasias based upon congenitally defective mesenchyme).

There was a time when otosclerosis "existed" only when clinically manifested, which was usually about the period of puberty or later. Since temporal bones in large numbers, from individuals tested before death, have been sectioned, it has been discovered that typical otosclerosis may exist with no impairment of hearing. Furthermore, typical otosclerosis has been found during the first year of life. The hereditary factor in most cases is now commonly accepted. To repeat, the involvement is usually bilateral and identical. For these reasons it would seem more probable that otosclerosis is due to some inherent peculiarity in the original mesenchyme, influenced by local morphology, rather than to a postfetal localized irritation. It seems rather significant that a number of cases of osteogenesis imperfecta have shown typical otosclerotic involvement of the aural capsules, since it is an accepted fact that osteogenesis imperfecta is due to an abnormality of the embryonic mesenchyme.

The literature has been flooded with reports of cases and theories of endocrine relationships. This is not surprising when we think of the influence upon general metabolism exerted by the thyroid and other glands of internal secretion; the influence upon growth of bone by the anterior lobe of the pituitary; the frequency with which otosclerosis manifests itself clinically, first at puberty, when there is a tremendous acceleration of bone change; the fact that pregnancy, during which the body is flooded with anterior lobe pituitary hormone, is a period when otosclerosis may either make its appearance clinically or, if present, show marked change for the worse; the fact that at the menopause, another

period of endocrine upheaval, otosclerosis may grow worse; the fact that the parathyroids control calcium metabolism, which is so important in the bone changes observed in otosclerosis, etc.

Despite the relationship between the glands of internal secretion and otosclerosis, it is safe to say that a scientifically proven, etiologic relationship has not yet been demonstrated.

Although classical states from endocrine disturbance, including marked obesity from posterior lobe pituitary insufficiency, obesity and myxedematous states from hypothyroidism, early senescence with loss of libido, weight and hair from decreased function of the anterior lobe of the pituitary, exophthalmic goiter from increased thyroid activity, are commonly seen, these individuals do not, as a rule, manifest signs and symptoms of otosclerosis or, when otosclerosis does occur in such cases, it is so infrequent that one is tempted to look upon the occurrence as coincidental.

There can be no doubt that where otosclerosis exists, the progress of the malady is influenced by the internal secretions. When one reads reports of improvement in hearing and decreased tinnitus, however, resulting from endocrine treatment as well as other forms of therapy, one is tempted to doubt the familiarity of the reporter with the histopathology of true otosclerosis. These case reports are practically all of individuals of puberty age or older. The underlying abnormality, the author believes, is mesenchymal and embryonic or it develops soon after the fetal period. The bone changes start as soon as ossification of the capsule is complete or possibly at an earlier period. Now when one visualizes the resorption spaces filled with fibrous marrow and partially obliterated by a new inferior bone, destruction of the annular ligament with ankylosis, more or less complete, etc., it seems rather futile to anticipate a *restitutio ad integrum* or anything approaching it from any form of therapy.

It would seem that if anything is ever to be accomplished therapeutically a diagnosis will have to be made during the intrauterine existence of the individual and treatment directed through the medium of the mother. This is not as farfetched as it may seem, for we know already that to avoid certain other disorders in the infant the mother must be carefully watched and treated for endocrine disease, vitamin deficiency, etc.

Until we know the exact nature of the mesenchymal cell abnormality and the period when secondary activity of these cells first manifests itself, therapy, it would seem, must remain but a futile gesture. However, it is not the desire of the writer to discourage further therapeutic experimentation.

In the chapter on the theory of regression will be found the intriguing conception of intra-embryonic conflict between genes for normal characters and the inhibited genes for regressive hereditary characters. In otosclerosis the genes for normal formation of the capsule suppress only temporarily the regressive genes of otosclerosis. Who knows but that some day, in some way (radium, X-ray, parenteral suffusion with actinic ray chemicals) these regressive genes will be permanently stilled in the developing embryo, through the medium of the mother.

THE REGRESSION THEORY OF OTOSCLEROSIS.

Prof. C. Emery,[11] in speaking of the investigation of certain anomalies of the human body in relation to atavism, states: "Genügt die Reihe der Säugetiere nicht, so geht es bis zu den Reptilien oder sogar zu den Fischen weiter abwärts." Before atavism came into fashion one spoke of animal resemblances.

The author tells of his daughter, who in early childhood had a definite copper-red tint to her hair. This later disappeared entirely. Neither the writer, his wife nor their parents or grandparents had had red hair. A sister of the author had had red hair in childhood. It was in the family history that somewhere far back red hair had existed.

Frequently children's eyes change color. The writer's six-year-old daughter had blue eyes which later changed to brown. The writer's eyes are blue, those of his wife are brown. The phenomenon observed in his daughter represents a struggle between the genes for brown iris, which were in the beginning less strong than the genes for blue iris. Such fleeting signs of latent heredity, he believes, are much more common than one thinks and are simply not observed in most cases. In these instances one is observing heredity of few generations; but they are really no different from other instances in which are manifested regressions representing conditions dating back geologic periods.

As an example, the writer selects a case, examined by himself, of hyperdactylia in a pig embryo. The cartilage which corresponds to the trapezium of the normal wrist (and possibly containing a rudiment of the first metacarpus) was unusually long and had the form of a metacarpus. There followed a phalanx and then an indifferent skeletal anlage. It was not possible to picture what the ultimate appearance of the supernumerary thumb would have been had the pig developed further, but a case reported by Ercolani of a fully developed hog corresponded exactly with the author's case. The supernumerary finger was articulated just as a thumb should be, but the metacarpus was blended with the trapezium and there were three phalanges as in the index finger.

The author looks upon his case and Ercolani's as examples of atavism, not because he believes that artiodactyla must have necessarily possessed a thumb but because, in the normal embryonic extremity of the pig, an indifferent, temporary anlage of the thumb bones appears. Its proximal division forms the trapezium, while the distal portion disappears entirely. The germinal tissues in the two cases hold as hereditary characters the preeocene elements to form thumbs. These elements exert only temporarily a slight influence upon the course of normal ontogenesis.

Emery reminds us that an anomaly may be looked upon as an atavism only when in the normal ontogenesis a trace of the formation appears. A formation of which there is no trace in normal ontogenesis may resemble some formation appearing in a lower form of life but cannot be considered to have been determined by an inherited germinal anlage. He mentions Rosenberg's report of the occurrence in man, of supernumerary upper incisors in three places, which phenomenon Rosenberg looked upon as an atavism pointing to the reptile stage of phylogenesis. Emery does not agree. He believes the condition resembles one of an early phylogenetic period but that the germinal inheritance is lacking; that the supernumerary teeth, having accidentally developed in the incisor region, naturally turn out to be incisor-like.

A completely latent heredity during countless generations ("sleeping germinal cells") which here and there manifests itself

through the development of a complex tissue structure cannot be accepted as a latent heredity without a most scientific proof.

A completely proven example of such a latent heredity are cases of dimorphism and metagenesis. The butterfly, under abnormal temperature conditions, will breed in different colors. The genes for these new colors are, under normal conditions, suppressed by the genes for the usual coloring. This phenomenon represents an artificially induced throw-back. Seasonal dimorphism is also related to the above phenomenon. In cases where abnormal temperature fails to produce the new color in breeding, the latent hereditary color gene is no longer present.

Emery states that the biogenetic basic laws appear to him in a new light, namely, the growing organization passes through a definite series of developmental stages, because (1) certain stages of the entire organism, as well as of the individual parts, are a mechanical necessity—that is, their form and sequence are determined by physicochemical laws, entirely independent of heredity and phyolgenesis; (2) the appearance or nonappearance of hereditary ancestral stages depends chiefly upon the presence or absence of the corresponding, determining genes in the germinal tissues. Such genes may date from times primeval or have originated in any intervening period. Weismann[12] states that the determining elements are in conflict during the development of germinal tissues and that within the same a natural selection occurs, the result of which is a simplifying of the germplasm by the suppression or destruction of certain anlages in favor of others more useful to the organism. Possibly the suppressed genes, representing rudimentary characters, start development more slowly and later than the more useful ones.

The study of embryologic stages often reveals fleeting anlages which represent the last traces of primeval heredity which no longer appear in the fully developed organism or in any living organism. As an example of this, Emery mentions the appearance, in mammalia, of prelacteal dental anlages.

Gray[13] states: "Apart from the hereditary tendency, there is no single cause to which otosclerosis can be attributed. A review of the evolution of the organ of hearing in the vertebrates shows

that the semicircular canals and vestibule reached their full development in the fish and made no further advance. The oval and round windows and the ossicles, as well as the cochlea, however, have all been evolved certainly since the first appearance of the amphibians, and possibly may have had no existence before the appearance of the reptiles. These structures therefore have been evolved at a period of time measured in millions of years later than the vestibule and canals." Gray is of the opinion that otosclerosis is a variation in the biologic sense of the word. Variations are apt to occur in structures that are of comparatively recent origin, and this would explain the fact that otosclerosis involves the footplate of the stapes, the bony walls of the cochlea and in some cases the nerve structures of the latter, while the other portions of the labyrinth are almost invariably untouched. If otosclerosis is a variation in the biologic sense, it may arise spontaneously and yet be transmitted, so that this also explains why, although a number of cases are obviously hereditary, there are others in which the affection arises apparently *de novo*.

In 1921, the same author stated: "The logical conclusion to be drawn is that the basic cause of otosclerosis is to be found in the organ of hearing itself and also that this condition exists in the organ of hearing of certain individuals only."

Although Gray emphasizes the fact that the vestibule and canals appeared long before the oval and round windows, it evidently did not occur to him that the closure of these windows in otosclerosis might represent a regression to the period when no windows existed. His belief that the condition can arise spontaneously is not at all proven by the fact that cases are seen in which no history of deafness in the family can be obtained; for it may well be that the trait has been dormant for generations. Further, there exist, without doubt, many cases of otosclerosis which never lead to ankylosis or nerve degeneration and therefore no deafness occurs to acquaint the family with the presence of otosclerosis. In such cases the secondary mesenchymal activity has probably met with some inhibiting influence or an anomalous involvement may have occurred, away from those parts concerned with the function of hearing.

Josef Fischer[14] reports signs of regression in a temporal bone from an individual of 28 years. From the anterior edge of the macula utriculi there arose toward the endolymphatic space a structure with an outer and inner epithelial covering. It extended along the upper and outer wall of the utriculus. The epithelial covering blended with the walls of the utriculus, dividing the utriculus into two sacs. The central portion showed nerves and blood capillaries. The median epithelial layer corresponded completely with the macular epithelium, showing hair cells, hair processes, etc. The lateral epithelial layer showed on the base also hair cells. Peripherally were high cylindric epithelia covered with a cubical type. The accessory structure showed also two cones: the inferior was long and showed a continuation of the macula epithelium; the lateral pointed outward and showed high cylindric epithelia, hair processes and a cupula. In a sinus utricularis was an atypical epithelial formation in the form of a sac. The epithelium of this sac showed hairs which were extremely long and similar to the neuroepithelial area in reptiles. On the upper median wall of the utriculus was a richly pigmented process covered with cuboidal epithelium and fine branching blood vessels. The crista horizontalis looked like a mammalian animal crista. It showed over the hairs, globular inclusions. The tendon of the tensor tympani at its origin was globular, as in mammalia. This the author looked upon as a sign of atavism. The lamina spiralis ossea with the ligamentum spiralis extended over the whole width of the floor of the vestibule as a thin bony plate. The membrana tympani secondaria arose from the lamina spiralis ossea. In some places the lamina spiralis ossea looked bifurcated and very large. This again was looked upon as a sign of atavism. The chief abnormality, Fischer looks upon as a third macula. He reminds us that in comparative anatomy we find a third macula in the pars inferior (lagena). This third macula he looks upon as either an advanced development with a better power of orientation or as an atavism (regression to the period of lagena).

Alexander,[15] in 1909, maintained that otosclerosis is a distinctly hereditary affection, but the characteristic clinical signs do not develop until about the time of puberty. The anatomic findings in the author's case of congenital deafness showed the otosclerotic

bone changes to be intra-embryonic. The author believed that the otosclerotic patient, even as a child, as a newborn infant, even as a fetus, shows the strongly vascularized bone foci in his petrous bone.

Gradenigo,[16] in 1887, stated that "sclerotic otitis" is not uncommonly hereditary; has as a particularly constant pathologic feature, lesions of the labyrinthine windows and especially ankylosis of the stapes. These characteristics indicate that it is a pathologic condition of embryonic, congenital origin closely related to the morphology of the ear.

Davenport,[17] of the Carnegie Institute of Genetics, in an analysis of the genetic factors responsible for the development of otosclerosis, concludes: "The hypothesis of two dominant genes is not negated in any of the groups of matings. The hypothesis that otosclerosis depends on a single dominant factor, on a single recessive factor, on a sex-linked recessive and a dominant autosomal gene, on two recessive genes one (sex-linked) and one a dominant recessive and a recessive autosomal gene meets with difficulties in application. The conclusion is reached that the simplest formula that approaches agreement with the findings is that of two dominant genes, one of which is sex-linked."

Taniyama[18] attributes the spongification in otosclerosis to the presence of cartilage rests in the bony capsule. On the border of the oval window he found cartilage remnants in individuals of 6 months, 3 years, 6 years and 12 years. Even in specimens from individuals of 20 and 54 years he could demonstrate cartilage rests in the same region but not clearly.

Jannuzzi[19] determined the blood groupings in all available members of five families, in each of which there were a number of otosclerotics. He found that all the deaf members of a given family belonged to the same blood group. In one family all were in group 2; in another, all were in group 4; and in the remaining three families, all of the deaf members were in group 3. The author considered that these data confirm the presence of the hereditary tendency in otosclerosis.

Kosokabe[20] was the first to show that the circumscribed cartilage remnant in the region in front of the oval window occurs more frequently in females. This may explain the more frequent

occurrence of otosclerosis in the female, in the sense that this circumscribed cartilage remnant may be regarded as the "germ" of the tumor-like otosclerotic process.

EXPLANATION OF REGRESSION IN OTOSCLEROTICS.

The ear, primeval, was probably a simple affair: a mere vesicle formed from an invagination of skin (ectoderm) and containing a calcareous deposit (otolith). The vesicle was connected with the primitive brain by means of ganglion cells and short nerve fibers. The sole function of the otic vesicle was to inform the organism of its position in space and to act as a receptor for certain vibrations. As movement occurred the epithelial cells lining the vesicle were stimulated by the otolith and the message was transmitted to the brain. There was no hearing function as we understand it, but the organism probably reacted to certain vibrations in the water.

As time went on and the movements of organisms became more complex, a more accurate organ of equilibration became necessary. There developed first one semicircular canal as an evagination or pinched off portion of the primitive vesicle. The canal was supplied with a nerve of its own. Later a second and finally a third canal developed. This made a very efficient organ of equilibrium; but no hearing function existed—only the sensing of vibrations in the water. Later the vesicle constricted into two sacs; one (utriculus) which communicated with the canals, the other (sacculus) smaller than the first and communicating with it. Both sacs were supplied with nerves. In the course of time an evagination (lagena cochleæ) from the sacculus made its appearance. The lagena had its own nerve ending. Probably the appearance of this forerunner of the cochlea marked the advent of a function more nearly related to hearing.

Not until much later did there appear an oval and then a round window, ossicles, etc.

Now, when the human embryo reaches a certain stage of development, certain ectodermal cells in the wall of the hind brain begin to proliferate to form the auditory placode. These cells are specifically designed to form the placode just as other ectodermal cells are destined to form the olfactory placode and nothing else.

Quickly the auditory placode sinks inward, closes off and becomes the otic vesicle, embedded in mesenchyme. We have then a reproduction of the ear primeval; a repetition of a process countless millions of years old. If no further development of the ear occurred the individual would be manifesting a regression to the most primitive aural structure. As normal development proceeds the organ of hearing and equilibrium goes through each stage of phylogenesis, so that a throw-back could result from cessation of development at any point or from a dystrophic return to a previous period, after normal development had been accomplished.

The otic vesicle, embedded in condensed mesenchyme, is said to be the sole factor in the stimulation of these mesenchymal cells to further proliferation and differentiation. Experimentally, an otic vesicle, transplanted to another region, will develop a capsule out of mesenchyme. The complex aural capsule with its many forms of bone, its windows, etc., would seem to be the end-result of specific mesenchymal cell activity with an inherent chromosome predetermination: however, this may not be the case; the noted complexities may result from stimulation by the multiformity and cell specificity of the ectodermal membranous labyrinth or from inhibitions from surrounding tissues. For example, the protrusion into the tympanum of Reichert's cartilage with its stapedial ring, which comes into apposition with the capsule, may exert an inhibition upon the capsule, for the transformation of mesenchyme into cartilage does not occur under the stapedial ring until long after the capsule is of pure cartilage. Again, cartilage formation may be inhibited at the internal meatus by the nerves which enter the labyrinth. Thus far, the mesenchyme cells need not possess a specificity to account for the morphology of the capsule. When we try to explain, however, the formation of the round window, it seems that we must fall back to the theory of specificity, for neither on the tympanal nor labyrinth side is there anything to account for the fact that differentiation into cartilage stops short where the round window with its membrane will be located. Since one cannot fairly make one rule for the internal auditory meatus and oval window and another for the round window, it would seem logical to conclude that the capsular

mesenchymal cells are different from other cephalic mesenchyme cells and are specifically destined to form the otic capsule with its windows, etc.

Now, in bone pathology (for example, in osteodystrophia fibrosa) it is a well known fact that when resorption of bone occurs there is a filling in with the basic substance of the bone-forming system, namely, mesenchyme. Further, that progress toward repair may be halted at any given point so that, instead of the reconstruction of the original type of bone, there may occur a regression to an inferior type. In such a case we must presume that, in the absence of interference, the basic material (mesenchyme) of the bone-forming system would have gone on to the ideal end-result, namely, the re-establishment of the proper type of bone for the particular locality. In bone pathology, the interference may be due to some generalized or local metabolic abnormality or to ontogenetic periodicity, etc. In the development of the otic capsule not all of the embryonic mesenchyme is differentiated into precartilage, cartilage and bone or into fiber "nucleus" and fiber bone. One of the characteristics which differentiates the otic capsule from other bones is the persistence of cartilage elements in the enchondral division, over the rim of the oval window, the footplate of the stapes and in the lining of the fissula ante fenestram, etc. These cartilage rests, where they have not yet undergone calcification, are nothing more than mesenchymal cells which have thrown out coalescing protoplasmic processes and become surrounded by matrix. In addition, it will be shown that there also persist, in various parts of the capsule, mesenchymal cells in their original form, after ossification is completed.

It is the theory of regression that the persisting mesenchymal cells retain a potentiality for differentiation and that in otosclerosis they are activated by the regressive trait of forming capsule as it is formed in lower vertebrates with no oval or round window. The proliferation of these cells of regression starts after the inhibiting normal mesenchymal elements have expressed themselves in the construction of the capsule. Like cancer cells, they have the power of proliferation and invasion (probably, chiefly through newly formed vessels). The cartilage rests disappear sooner or

later in otosclerosis. They manifest less resistance to the invading mesenchymal elements than does the bone of the capsule. Invasion of the osseous structure is probably initiated by the products of cartilage degeneration plus interference with circulation: there follows also, in some cases, osteoclastic activity; in all cases vessel invasion. The resorption areas are quickly filled by proliferating mesenchyme, which is transformed into a mass of fibroblasts surrounding newly formed vessels. There follows peripheral fiber formation and calcification, the end-result being the formation of an inferior type of bone (fiber-bone), characterized by irregularity of cells and fibers, and theoretically rich in cementum, accounting for the blue stain; although iron in the focus may be the reason. (See Hagen's abstract in this work.)

The cartilage covered footplate is encroached upon from the fissula region and finally blends with the capsule from which it was derived. The round window may show the same involvement. Where cartilage rests are less common (canal region) otosclerotic changes are not so frequently seen. However, all persisting undifferentiated mesenchymal cells throughout the capsule of predisposed individuals probably contain the genes of regression and are therefore capable of forming a focus. When sufficiently inhibited by genes of normal development no focus of otosclerosis develops but transmission to progeny may occur.

SUMMARIZING.

The capsule of the labyrinth is developed from a condensed mesenchyme which differentiates on the one hand into fiber "nucleus" and fiber-bone; on the other, into precartilage, then cartilage and bone. Cartilage elements persist in certain localities, after ossification is completed. Certain lower vertebrates of today possess otic capsules without windows and with only utriculus and canals. Phylogenetically there was probably a period when oval window, round window, cochlea and sacculus had not evolved. In certain individuals there exists a transmitted regressive trait which leads to a secondary embryonic mesenchymal activity with invasion of cartilage and then bone, and resulting in a wild proliferation of mesenchyme, a differentiation into fibroblastic tissue and finally fiber-bone. The oval window with the footplate derived from the capsule and nonexistent in certain lower verte-

brates is obliterated; the footplate returns to the tissue whence it was derived. In the same way, but less frequently, the round window, internal auditory meatus and canal region may show the effect of the secondary mesenchymal activity.

The fact that otosclerosis is usually bilateral with identical involvement speaks for heredity and emphasizes the importance of the morphology of the predilection site. In certain lower vertebrates the two ears are essentially one organ, the utriculus of one being connected with that of the other by means of canals above and below the brain (possibly evolved from the lateral-line organs which are joined by transverse canals).

The fissula ante fenestram, in which otosclerosis often begins, is a vestigial structure which may show cartilage throughout life and is therefore predisposed to mesenchymal invasion. The primary involvement being of a vestigial structure suggests its regressive character. The homologue of the fissula is the canalis fenestræ ovalis of the frog.

The question may arise as to why the secondary mesenchymal activity in otosclerosis results in the formation of fiber-bone and not cartilage, the capsular structure of the lowest vertebrates. The answer is that otosclerosis begins after ossification is completed or nearly completed: at a time when differentiation into cartilage is no longer ontogenetically possible. The formation, in man, of cartilage "nucleus" occurs in the capsule before the eighth week and never by subsequent dystrophy.

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REFERENCES.

1. Hagens, E. W.: "Otosclerosis." Archives of Otolaryngology, Vol. 13, No. 6, page 824-828, June, 1931.

2. Brunner, Hans: Wiener Klinische Wochenschrift, No. 6, February 6, 1931, pages 173-176.

3. Leiri, F.: Acta Oto-laryng., Vol. XVI, Fasc. 1, p. 57-71.

4. Dickie, J. K. M.: Archives of Otolaryngology, July, 1931, Vol. 14, No. 1, pp. 86-93.

5. Lange, W.: Ztschr. f. Hals-Nasen-u-Ohrenh., 25:1, 1930.

6. Weber, Moritz: "Bone Pathology and Its Relation to the Problem of Otosclerosis." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. XXVII, No. 4, pp. 1232-1256, December, 1928.

7. Weber, Moritz: "Osteodystrophia fibrosa, ihre Pseudotumor und Cystenbildende, juvenile, hyperostotisch-porotische Form beim Hunde." Zugleich ein Beitrag zur Frage ihrer Pathogenese und Ätiologie. Beiträge zur Path. Anat. u. zur allg. Path., Bd. 82:1929.

8. Weber, Moritz: "Otosclerosis in Its Histogenic Relations to Osteodystrophia Fibrosa (Ostitis Fibrosa)." Archiv. of Otolar., January, 1930, Vol. II, pp. 1-35.

9. Weber, Moritz: "Zur theorie der experimentellen Otosklerose. February 1, 1930. Zeitsch. für Hals, Nasen-u-Ohrenh., Band 26, Heft 1, pp. 62-105.

10. Weber, Moritz: Zeitsch für Hals-Nasen-und-Ohr., Band 28, Heft 4, pp. 415-450, July 20, 1931.

11. Emery, C.: "Gedanken zur Descendenz-und Vererbungstheorie: Homologie und Atavismus im Licht der Keimplasma-Theorie." Biologisches Centralblatt. April 1, 1896. Band XVI, No. 7, pp. 344-352.

12. Weismann: "Neue Gedanken zur Vererbungs frage: eine Antwort an Herbert Spencer." Jena, 1895.

13. Gray, A. A.: Textbook, "Otosclerosis," 1917 (Abstract from p. 254, Vol. I. Otosclerosis, Amer. Otol. Society).

14. Fischer, Josef: "Eine bisher nicht beobachtete Missbildung im häutigen Labyrinth." (Macula III.) Zeitsch. für Hals-Nasen-und-Ohr. Band 27, pp. 582-585, 1930.

15. Alexander, G.: Quoted on p. 245 of Vol I, "Otosclerosis," Amer. Otol. Society.

16. Gradenigo, G.: Quoted on p. 270 of Vol. I, "Otosclerosis." Amer. Otol. Society.

17. Davenport, Charles B.: "Genetical Study of Otosclerosis." Amer. Otol. Soc., Abstract Archiv. Otolar., September, 1932, Vol. 16, No. 3, p. 438.

18. Taniyama, A.: Quoted from p. 275, Vol. I, "Otosclerosis." Amer. Otol. Society.

19. Jannuzzi, S.: Arch. Ital. di otol., 40:499, 1929 (Ref. from Dickie, Loc. Cit.).

20. Kosokabe, H.: Quoted from Vol. I, "Otosclerosis." American Otol Society, p. 296.

XCIV.

Neoplasms of the Nose, Throat and Ear.

CARCINOMA OF THE ESOPHAGUS.*

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Carcinoma of the esophagus presents one of the most melancholy chapters within the realm of medicine. The mortality is practically 100 per cent, with only a few lasting cures reported by isolated surgeons, namely, Torek's[1] case, dying of pneumonia thirteen years after operation; Garre's[2] case, alive twelve years after operation, and Turner's case, alive ten years after operation. Gluck's[3] brilliant results, exhibited by his operation of total laryngectomy for carcinoma of the larynx, stands out in prominent contrast to his six surviving cases out of 114 operated upon for malignancy of the esophagus. The relative anatomic inaccessibility of the disease is obviously the cause of the difficulties in both diagnosis and treatment with which this paper will deal.

Perhaps our teaching has been at fault. The textbook picture of carcinoma of the esophagus usually presented, consisting of cachexia, emaciation and extreme dysphagia, is a terminal one. More emphasis should be placed upon the symptoms of cervical or retrosternal discomfort in swallowing, that either recurs or tends to persist. This symptom to us is an absolute indication for esophagoscopy, preceded by fluoroscopy with the barium meal. Roentgenology is utilized by us, mainly as an adjunct to esophagoscopy, namely, to rule out extra-esophageal pathology causing pressure from without, as well as the one important contra-indication to endoscopy—aneurysm of the aorta. Esophagoscopy is performed, whether roentgenologic evidence of esophageal dis-

*Another of the series of articles by Drs. Beck and Guttman on Neoplasms of the Head and Neck. When the entire series will have appeared in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, they will be published in monograph form.

ease is present or not; first, to obtain a biopsy and ascertain the nature of the pathology, and secondly because clinical experience has shown that X-ray signs are not absolute, as about 10 per cent of the cases returned with the X-ray diagnosis of carcinoma of the esophagus have been erroneous and from 10 to 20 per cent of the cases returned as negative have been found to have endoscopic evidence of pathology. Undoubtedly this percentage of error must increase as more and more earlier cases are examined.

The esophageal bougie is only mentioned to be condemned; indeed, Jackson sounded its death knell with his famous aphorism that, "As a diagnosis instrument it is always negative early, inconclusive later, ineffectual at best, and often fatal"—the last referring to the not infrequent perforation of the esophagus with a subsequent mediastinitis and death. To repeat: the absolute diagnosis of endo-esophageal carcinoma rests on esophagoscopy and biopsy. Indeed, we feel that this condition can only be discovered early and with absolute certainty by endoscopy.

Endoscopically the appearance of the esophagus depends upon the location of the growth, whether endo- or peri-esophageal, and the stage of the growth in the period of its existence. Early endo-esophageal malignancy may appear as a small erroded nodule, no larger than a split pea; later, the nodule grows while the eroded epithelium changes to frank ulceration which subsequently fungates. The picture of a bleeding, ulcerated, fungating endo-esophageal mass is the most common lesion seen. Peri-esophageal carcinoma is less frequent, forming less than 10 per cent of the cases. Early it may present as a red, firm and infiltrated area within the esophagus wall, palpable with the tube mouth as a hard mass. Later rupture into the esophagus may occur and a picture similar to endo-esophageal carcinoma may be seen. A biopsy is performed on any pathology encountered, although we question the advisability of biting through normal mucosa when confronted by an area of infiltration.

From a pathologic point of view, surgery should show more favorable results, especially when cases are seen early. Helsley[4] has found that 64 per cent of seventy cases of carcinoma of the esophagus studied at autopsy presented a lesion that was confined to the viscus, and Blauwkwip[5] found this to be the case

in 49 per cent of 125 cases studied postmortem. These are certainly favorable facts from a gross anatomic point of view when one considers that these cases practically all died from this dire affliction. There is, however, an unfavorable element. Vinson and Broders,[6] in a histologic study of esophageal carcinomata, have found a high proportion to be anaplastic and therefore highly malignant growths. These types, which some pathologists have called transitional cell carcinomata, are highly radio-sensitive and in all probability are best treated by irradiation. The less malignant forms of the so-called squamous cell carcinoma and adenocarcinoma should be more amenable to surgical treatment.

The treatment of carcinoma of the esophagus has, in the main, been of a more or less palliative nature. Measures are instituted most frequently to maintain a patent esophageal tract with the idea of relieving dysphagia. Early neoplasms located in the cervical portion are, however, open to surgical attack with a minimum immediate mortality as compared to growths of the thoracic portion.

Carcinoma located at the level of the larynx when operated upon early offers the best chance of any for lasting cure, after the method of Trotter and Orton.[7] If the lesion is located in the posterior aspect of the esophagus, lateral esophagotomy is performed and the esophagus resected, leaving only the anterior portion forming the party wall between the larynx and the esophagus. The esophagus must be resected wide of the growth. A nasal feeding tube is inserted via the nose and transmitted by the esophageal defect and a large flap of skin is turned in to make good the defect in the posterior portion of the esophagus. Later the opening in the neck is closed by a sliding flap of skin. If the lesion is located in the anterior portion of the esophagus, invading the larynx, the combined laryngopharyngectomy is performed and the posterior part of the esophagus left in situ. In these cases the skin flap is turned in to make good the defect of the anterior portion of the esophagus, and the opening closed at a later date by a sliding skin flap. In some cases sufficient esophagus is left, permitting closure without turning in a flap.

Carcinoma located in the cervical esophagus is best treated by a two-stage operation. In the first stage the esophagus is dis-

sected free on both sides of the neck and off of the trachea and mobilized. It is then surrounded by iodoform gauze for several days in order to seal off the mediastinum and at a later date the growth resected. Duff Allen[8] advocates an ingenious operation. During the first stage of the operation, after the esophagus has been mobilized, two layers of facia lata are placed around it. In the second stage, some time later, the external layer of the facia lata is incised, and the internal layer resected with the esophagus, leaving the external layer filling the defect of the resected esophagus. Galloway[9] has described a method of attack for carcinoma of the cervical esophagus that employs surgical endothermy in the destruction and resection of the growth. We have been especially impressed with the possibilities of his method.

As we have had no experience with the surgical treatment of carcinoma located in the thoracic portion by either posterior mediastinotomy or combined abdominal and transpleural resections of carcinomata located at the cardia, no further discussion will be attempted.

Surgical diathermia via the esophagoscope has been utilized in the past few years. It is, of course, only applicable to endo-esophageal growths that are small and circumscribed. Peri-esophageal extensions of the neoplasm cannot be followed and, therefore, as far as a curative agent is concerned, it is more or less futile. In all probability this field of utility will be restricted towards restoring the lumen in some such manner as advocated by Wright.[10] He employs a special electrode with a flange disc mounted on an insulated shaft that has a bougie tip. The instrument is passed through the tract in the neoplasm beyond its distal portion and is then connected to a diathermy machine. The current is then turned on and the electrode slowly withdrawn, thus cutting a tract through the neoplasm the diameter of the size of the disc electrode. The use of various ball and pointed electrodes endoscopically, as advocated by Hesse,[11] in all probability will do no more than this.

Irradiation therapy is in the main palliative in nature. Guisez reports three cases of five years' cure with radium in esophageal carcinoma, but apparently no one else has been able to duplicate

his results. We have employed deep X-ray therapy, implantation of radon seeds, and contact with radium element, placed within the lumen in opposition to the neoplasm. These have always been followed by an increase in the esophageal lumen with less difficulty in swallowing. The improvement never lasted over a period of two months at the most. Repeated treatments were followed by increasingly less definite results until irradiation at length became ineffective. Undoubtedly this decreasing effect of irradiation was due to an increasing tolerance of the neoplasm to the gamma ray.

The radon seeds are implanted via the esophagoscope, using a special implanter, the needle of which has a guard preventing too deep introduction and perforation of the tissues. Well filtered seeds are advocated, containing one to two millicuries with a .3 mm. platinum wall. Four to six seeds are inserted about the circumference of the lesion, special care being taken not to perforate into the mediastinum.

Radium element, well screened by at least 2 mm. of platinum or its equivalent, may be placed against the neoplasm via the esophagoscope and suspended by means of a thread. Either a 12½ or 25 mg. capsule is applied every few days until a dose of 500 to 1500 mg. hours is effected. One of the most promising methods of irradiation is the use of a highly filtered four-gram pack of radium at a distance through four portals, anterior, posterior and lateral, from each side.

Intubation.—One of the most important palliative measures is the use of an intubation device preventing starvation and thirst. These may be of the rubber variety advocated by Jackson. Of late we have been more favorably impressed with the coiled wire tube devised by Soutar.[12] (Fig. 1.) Before intubation can be successfully carried out a more or less patent lumen must be present. The device is put into place under direct vision by esophagoscopy and not rarely a preliminary dilatation or irradiation may be necessary to provide a lumen. We are becoming more and more impressed that the best we can offer these unfortunate individuals, if surgery is not feasible, is a successful intubation. By this means starvation and thirst are eliminated and the patient permitted to expire without the disagreeable concomitant of a gastrostomy.

Fig. 1. Soutar intubation tube in the esophageal canal.

Dilatation.—We have discarded dilatation in the routine treatment of carcinoma of the esophagus and employ it only to provide a lumen for the placement of an intubation tube. When attempted it must be cautiously performed under visual guidance via the esophagoscope or over a swallowed thread in order to avoid perforation and possible mediastinitis.

Gastrostomy is indicated only if irradiation and intubation are ineffective in maintaining a patent esophagus.

CONCLUSIONS.

1. Cervical or retrosternal discomfort in swallowing should be an absolute indication for esophagoscopy, especially if the patient is within the so-called cancer age.

2. In the diagnosis of carcinoma of the esophagus, esophagoscopy is preferred to, and must be preceded by roentgenology, and a biopsy performed when pathology is encountered.

3. More may be expected from surgery in carcinoma of the esophagus if the lesion is diagnosed early and is located in the cervical portion.

4. Irradiation, whether by the so-called radium bomb, deep X-ray therapy, radon seeds or the actual radium element, is mainly palliative and aids in maintaining a patent esophageal lumen.

5. Intubation is an important palliative measure that makes the patient's existence easier by preventing starvation and thirst and thus obviating gastrostomy.

6. Dilatation is utilized only as a preliminary step to intubation.

7. Gastrostomy is a measure of last resort and only utilized when irradiation and intubation fail to maintain a patent lumen.

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REFERENCES.

1. Thorek, F.: Surgery of the Esophagus. Nelson Loose Leaf Surgery, 1927, Vol. 4, p. 119, T. Nelson & Son, New York.

2. Garre: Resection des esophagus. Munch. Med. Wchnschr. April, 1898, p. 439, and May, p. 557.

3. Gluck: Problem und Ziele der Chirurgie der Oberen Luft und Spiesewege. Monatschr. f. Ohrenheilk., 1921, Vol. 55, p. 1150.

4. Helsley: Metastatising Tendency of Esophageal Carcinoma. Annals of Surg., Vol. 77, p. 272, 1923.

5. Blauwkuip: Cancer of the Esophagus. Abstract in Journ. Amer. Med. Asso., Vol. 83, p. 1630.

6. Broders and Vinson: Degree of Malignancy of Carcinoma of the Esophagus. Arch. of Otolaryn., Vol. 8, No. 79, 1928.

7. Orton: Lateral Transthyroid Pharyngotomy. Arch. of Otolaryng., Vol. 12, No. 3, 1930.

8. Allen: Experimental Reconstruction of the Esophagus with Autogenous Fascial Transplant. Arch. of Surgery, Vol. 10, p. 374, 1925.

9. Galloway: Carcinoma of the Cervical Esophagus. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. XL, No. 2, 1931.

10. Wright: Carcinoma of the Esophagus. Brit. Journ. Surgery, Vol. 15, No. 71, 1927.

11. Hesse: Endoscopic Electrocoagulation of Carcinoma of the Esophagus. Zeitsch. f. Hals. Nas. Ohr., Vol. 27, No. 3, p. 517.

12. Soutar: Treatment of Carcinoma of the Esophagus. Brit. Journ. Surg., Vol. 15, No. 76, 1927.

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XCV.

IMPERMEABLE STRICTURE OF THE ESOPHAGUS RELIEVED BY RETROGRADE BOUGINAGE WITH THE AID OF A CYSTOSCOPE INSERTED THROUGH A GASTROSTOMY.*

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CINCINNATI.

The treatment of impermeable cicatricial stricture of the esophagus presents an extremely difficult problem. Fortunately, such complete strictures are comparatively rare and are most apt to occur in neglected or improperly treated cases. In a tabulation of 137 cases of cicatricial stenosis at the Vienna Clinic, Heindl[1] reported six as impermeable.

Complete atresia of the esophagus is characterized by the retention and regurgitation of all food taken by mouth, by the drooling of saliva and by the rapid emaciation and dehydration of the patient. Fluoroscopy and the X-ray film show the ingested barium, or preferably the lipiodol (Iglauer),[2] to be arrested at the site of the occlusion, and no traces of the opaque substance are visible below the obstruction. In the presence of a gastrostomy not even the faintest traces of swallowed chemicals can be recovered from the stomach. Esophagoscopy shows the esophagus completely occluded by cicatricial tissue through which it is impossible to pass a filiform bougie or force air upon ballooning the esophagus.

TREATMENT.

In the treatment of impermeable stricture there are two avenues of approach, either by external operation or by endoscopic methods. In this paper only the latter will be considered, but it should

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be stated that external operation should never be undertaken until all endoscopic methods have failed. The chances of restoring the lumen are more favorable in a short stricture without marked angulation at the site of occlusion.

The stricture case usually presents himself with a gastrostomy. If the stomach has not been opened and if the esophagoscopist is unable to re-establish the lumen at the time of his first examination, there should be no delay in having a gastrostomy performed. When possible, the gastrostomy should be so placed as to render the lower end of the esophagus easily accessible from the artificial opening into the stomach.

The stricture may be approached from above by peroral esophagoscopy, from below by retrograde esophagoscopy, or by a combination of both methods. In the presence of a stricture the esophagus is usually markedly dilated above the occlusion and has the shape of an inverted funnel below the obstruction. It follows that the thin walls above the stricture are easily ruptured by manipulations, while the thicker walls and the axial approach from below offer a safer and better prospect for relief by retrograde procedures.

Various methods have been employed in restoring the lumen of the esophagus. In 1911, Mosher[3] reported a case of complete corrosive stricture which came under his care after numerous manipulations, including the introduction of a cystoscope into the stomach and attacking the stricture from below, had failed. Combined retrograde and peroral esophagoscopy also failed to relieve the stenosis. Finally, upon ballooning the esophagus from above and carefully picking the stricture apart with a small metal olive, the stricture was unglued and the patient eventually made a complete recovery.

In 1914, I[4] reported a case of post-typhoid atresia of the esophagus in which the late Dr. John W. Murphy and I succeeded in restoring the lumen by the use of combined peroral and retrograde esophagoscopy. We found that transillumination from one esophagoscope to the other aided us in finding our way. The stricture was burned through with the point of a long electrocautery. A bougie was then passed through the esophagoscope

and could be palpated under the gastric mucosa, evidently having dissected up the esophageal and gastric mucous membrane in its downward passage. The patient was removed from the hospital contrary to our advice, but began to swallow while on the homeward journey and continued to swallow up to the time of his death, which occurred about three weeks after the operation. His death was probably due to a mediastinitis.

King[5] reported a case, in 1924, from the Laryngological Department of the Cincinnati General Hospital, in which he employed combined peroral and retrograde esophagoscopy controlled by a monoplane fluoroscope. The stricture was picked open with a forceps; the patient made a complete recovery and gained 100 pounds in weight after the operation.

Jackson[6,7] has succeeded in restoring the lumen of the esophagus in several cases by combined peroral and retrograde esophagoscopy controlled by a biplane fluoroscope. One death occurred in his series. The biplane fluoroscope certainly renders the procedure safer and more accurate, but both Jackson and Tucker[8] point out the danger involved, and the latter estimates the mortality to be as high as 50 to 75 per cent.

The use of electrolysis has been advocated by several European authors, notably Guisez,[9] Lotheisen[10] and Heindl,[1] all of whom report cases of impermeable stricture which became patent after the application of the electric olive introduced through the esophagoscope.

Stricture at the lower end of the esophagus has been successfully treated by peroral esophagoscopy combined with tactile digital and instrumental approach through an opening in the stomach. Jackson,[6,7] Heindl[1] and Imperatori[11] each record such a case.

I should like to report a case in which an impermeable stricture was overcome by the introduction of a cystoscope into the stomach and the passage of a ureteral catheter up the esophagus.

Miss N. S., age 26, came under my care on December 18, 1931, complaining of inability to swallow anything.

Present Illness.—Sixteen months previously the patient attempted suicide by swallowing half a can of lye mixed with water.

She then tied a towel around her neck in an effort to prevent the caustic from coming up. She was in a hospital for eighteen days thereafter, vomited blood at this time, and was unable to swallow for three or four days. For the next six months she could swallow only part of the time. She then was admitted to the University Hospital at Columbus, Ohio, where on March 18, 1931, a gastrostomy was performed after the method of Weitzel. At this time a very narrow annular stricture of the upper portion of the esophagus was found and the esophagus was dilated through an esophagoscope about ten or twelve times by Dr. Beatty. While in the University Hospital the patient refused to swallow a string. She was discharged from the Columbus Hospital on September 7, 1931. For the past four weeks the patient has been unable to swallow anything. Her previous history is unimportant except that she had a "nervous breakdown" in 1928 and worried about herself.

Physical Examination.—The patient is well nourished and receives all her food through a gastrostomy. She expectorates about 1,000 cc. of saliva during every twenty-four hours. She is very nervous, almost hysterical at times, and complains of pain and a burning sensation between her shoulder blades and under the lower part of the sternum. The general physical examination was unimportant.

X-ray Examination.—On December 19, 1931, the patient was given lipiodol to swallow, and under the fluoroscope an obstruction was noted two or three inches down the esophagus, but the lipiodol was immediately regurgitated. The X-ray films showed traces of the oil, but also showed that none of the lipiodol had passed beyond the obstruction, which was at the level of the sternal notch.

Esophagocopy.—On December 21, 1931, an esophagoscope was introduced and a complete fibrous, slightly funnel-shaped stricture was found at a depth of seven inches from the upper incisor teeth. An attempt was made to pass the stricture with a flexible bougie but this was impossible. I also attempted to pick the stricture open with a forceps, but this also failed. An esophagoscope was introduced through the opening in the stomach, and an

attempt was made to reach the esophagus from below, but this also failed. On the following day Dr. Henry Freiberg introduced a cystoscope into the stomach, and a ureteral catheter (No. 7) was passed through the cystoscope into and up the esophagus until an obstruction was encountered. X-ray films were immediately taken and showed the tip of the catheter at the level of the fifth dorsal vertebra (Fig. 1). Iodopin (40 per cent) was then injected through the catheter and another X-ray taken, which showed the esophagus filled with the opaque oil below the stricture (Fig. 2). Some iodopin was then injected through a catheter inserted through the nose, and another film showed the upper limits of the stricture. By comparing the films the vertical extent of the stricture was seen to be about two millimeters.

Subsequent History.—On December 24, 1931, about forty-five hours after the passage of the ureteral catheter, the patient, while sucking some candy, suddenly noticed that she was able to swallow. She then took some water by mouth, and succeeded in forcing it into the stomach. Two days later she succeeded in swallowing a string, and retrograde dilatation with a soft rubber catheter was begun. This treatment was continued until January 1, 1932, at which time the patient was able to eat very well, with practically no difficulty in swallowing, and she was discharged from the hospital.

Subsequent Treatment.—Since that time the patient has been dilated at regular intervals by Dr. Williams of Middletown, Ohio. A No. 24 French catheter can now be easily pulled through the esophagus. She takes all food by mouth, has lost her nervousness and is in excellent physical condition.

COMMENT.

When the ureteral catheter was introduced through the cystoscope, it was our intention to determine whether it could be passed up the esophagus, and if so, we desired to determine the condition of the esophagus below the stricture. The roentgen film showed that the catheter had been arrested at the level of the body of the fifth dorsal vertebra. The injected iodopin, however, had reached the lower limits of the stricture and showed the contour

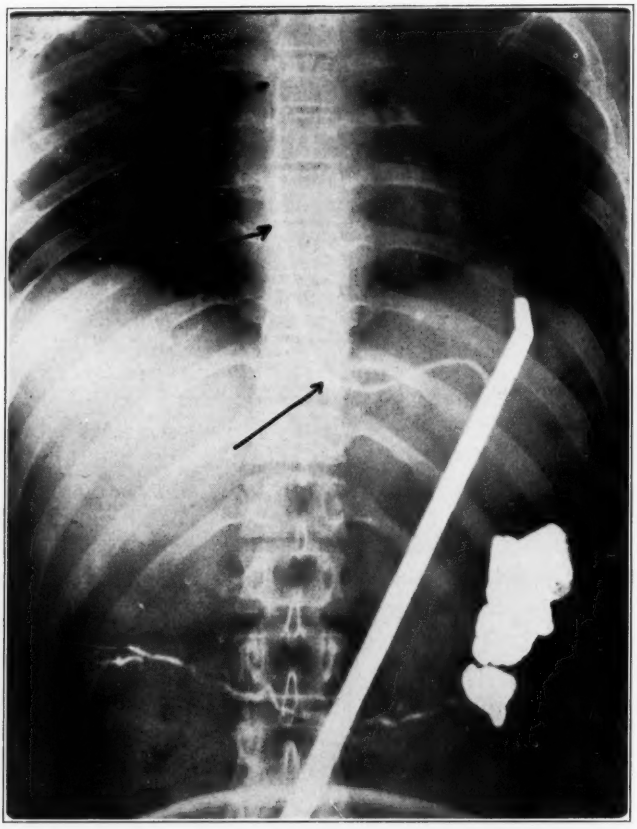

Fig. 1. Roentgenogram showing cystoscope in the stomach and a retrograde catheter introduced up the esophagus to the fifth dorsal vertebra.

of the esophagus below the stricture. By comparing the films we were enabled to determine the vertical extent of the stricture, which was but a few millimeters in thickness.

A few days after this examination we intended to reintroduce the ureteral catheter, and one of us was to push on the catheter while the other was to observe the stricture through the esophagoscope from above. In case any dimpling occurred in the stricture, it was our intention to burn through it with the point of a long diathermy electrode, to expose the tip of the catheter, and then to pull it through the stricture. Much to our surprise, the patient began to swallow on the second day after the retrograde bouginage, and later she succeeded in swallowing a string, which gave us the key to the situation. The introduction of the catheter probably opened some secondary strictures (Fig. 2) below the main stricture, and the injection of the oil must have further separated the walls of the esophagus. After the passage of the retrograde catheter, the entrance of air from the stomach into the esophagus was probably facilitated. These three factors, together with previous manipulations above the stricture, were probably all concerned with the sudden restoration of the lumen.

FURTHER EXPERIMENTS.

In order to determine whether retrograde bouginage through a cystoscope was feasible as a routine procedure, three other gastrostomized patients were examined in the same manner. The patients were aged four (Fig. 3), three and two years, respectively, and all three were under treatment for lye strictures. In each case Dr. Freiberg succeeded with the greatest ease in introducing the catheter up the esophagus and into the pharynx. At one time one of the children refused to swallow her string and a retrograde catheter was introduced, enabling us to attach a string and pull it down into the stomach.

INVESTIGATION OF A CASE OF CONGENITAL ATRESIA OF THE ESOPHAGUS.

A case of congenital atresia of the esophagus was recently admitted to the Surgical Service of the Cincinnati General Hos-

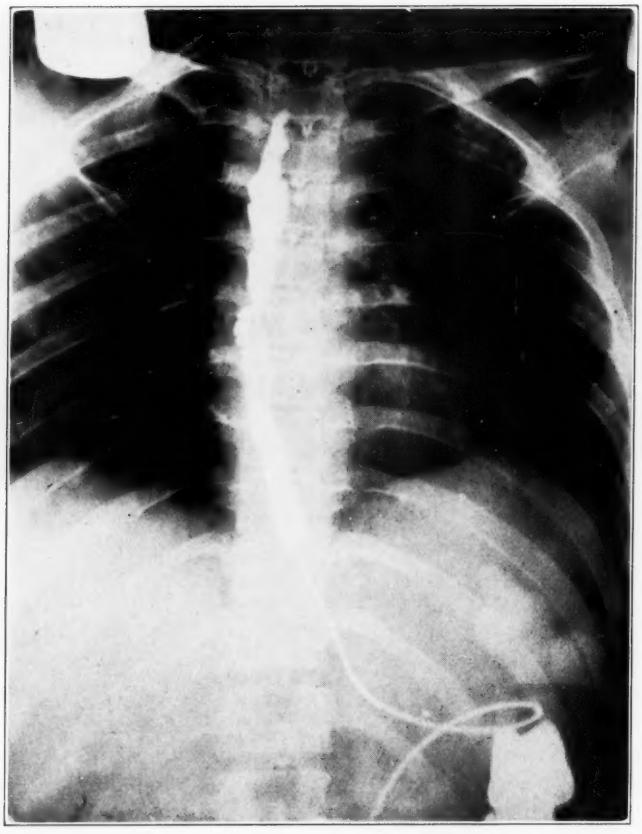

Fig. 2. The esophagus below the stricture is shown filled with iodized oil which has been injected through the retrograde catheter. The oil has reached the main stricture above. Several minor strictures and small diverticula can be seen in the esophagus.

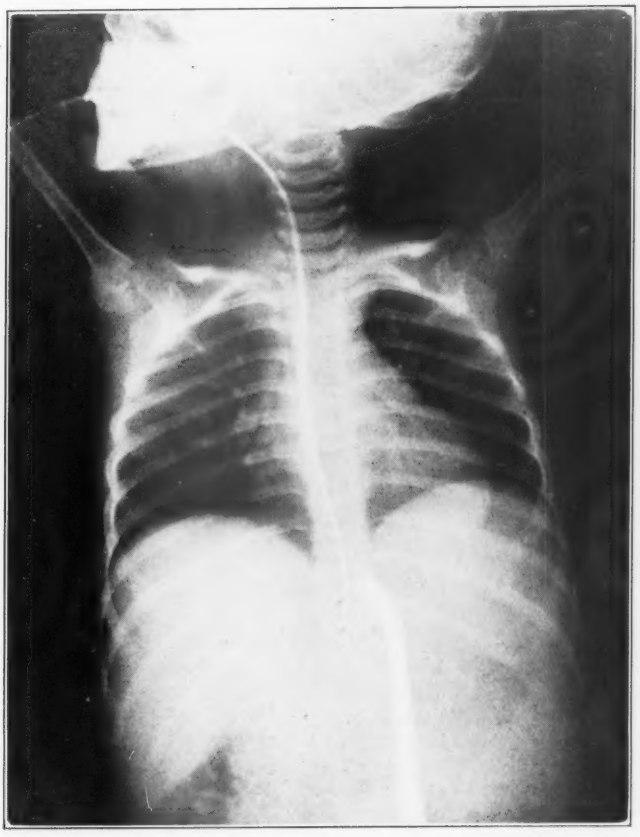

Fig. 3. Roentgenogram of a four-year-old child. A cystoscope can be seen in the stomach and a retrograde catheter has been passed up the esophagus into the pharynx and out of the mouth.

pital. The barium meal filled out a blind pouch of the esophagus terminating at the level of the fifth dorsal vertebra. A gastrostomy was performed. The infant died when it was ten days old. After death a ureteral catheter was passed up the esophagus from the stomach and lipiodol was injected through the catheter. The lipiodol filled the lower segment of the esophagus, from which it entered the trachea through an esophagotracheal fistula (Fig. 4). At autopsy all the X-ray findings were confirmed. In addition a massive cerebral hemorrhage was found. Dr. Mont Reid[12] has elsewhere commented on this case in some detail.

COMPARISON OF CYSTOSCOPY WITH RETROGRADE ESOPHAGOSCOPY.

Retrograde esophagoscopy is not always easy of accomplishment. The gastrostomy may be poorly placed, the folds of the stomach may get in the way, and the cardia may be difficult to find, especially since the retrograde esophagoscope exposes but a very limited field at any one time. The insufflation of air is a great aid, but may cause violent contractions of the stomach.

With the cystoscope in the stomach the entire cavity can be viewed with the greatest ease after the stomach has been filled with water. Pathologic conditions, such as ulcers, lye burns, or scars should be readily delineated. An expert urologist, once oriented, can pass a catheter up the esophagus within a very few minutes, and with no discomfort to the patient. If necessary, the procedure can be carried out under fluoroscopic control.

SUMMARY AND CONCLUSIONS.

1. Various methods of treatment of impermeable stricture of the esophagus are discussed.

2. A case is reported in which the atresia was overcome by the retrograde introduction of a ureteral catheter passed through a cystoscope.

3. Injection of opaque oil through the retrograde catheter enabled us to obtain an X-ray film of the esophagus below the stricture. This procedure, combined with the introduction of oil from above, demonstrated the thickness of the stricture.

Fig. 4. Postmortem study of a case of congenital atresia of the esophagus. A ureteral catheter has been passed up the esophagus from the stomach, and lipiodol has been injected through the catheter. Lipiodol filled the lower segment of the esophagus, from which it entered the trachea and bronchi through an esophageal tracheal fistula. The upper segment has also been injected from above with lipiodol.

4. Retrograde passage of a ureteral catheter was practiced on three other gastrostomized patients, proving the ease and feasibility of this procedure.

5. The retrograde introduction of a ureteral catheter should be of considerable value in the study and treatment of complete atresia of the esophagus.

6. Retrograde catheterization may easily be repeated any number of times if necessary.

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BIBLIOGRAPHY.

1. Heindl, A.: Klin. Beobachtungen an 137 Gutartigen Oesophagus-Stenosen. Deutsch Zeit. f. Chir., 198-199:252, Vienna, 1926.

2. Iglauer, S.: Nonopaque Foreign Bodies in the Esophagus and Esophagotracheal Fistula. Arch. f. Otolar., 7:229-233, March, 1928.

3. Mosher, H. P.: The Use of the Esophagoscope in Esophageal Surgery. Boston Med. and Surg. Jour., Vol. CLXV, 11:401-403, Sept. 14, 1911.

4. Iglauer, S.: Discussion of Jackson, C., Esophageal Stenosis, etc. Trans. Amer. Laryngol., Rhinol. and Otol. Soc., p. 211, 1914.

5. King, E.: A New Method of Restoring the Lumen in a Case of Impermeable Stricture of the Esophagus. Surg. Gyn. and Obst., 39:596, 1924.

6. Jackson, C.: Bronchoscopy and Esophagoscopy, Textbook. W. B. Saunders Co., p. 350, 1927.

7. Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Textbook, p. 1069, Phila., 1929.

8. Tucker, G.: Stricture of the Esophagus—Diagnosis and Treatment. Laryngoscope, June, 1931.

9. Guisez, J.: Diagnostic et Traitement des Rétrécissements Cicatricial de L'Oesophage. Arch. Internat. d. Laryn., D'Otol. et de Rhinol., 34-35: 112, 1912-13.

10. Lotheisen, G.: Zur Behandlung d. Oesophagus-Strikturen. Zbl. Chir., 50, 11:431-432. See also Wien Med. Woch., 72:144-7, 1922; and Wien Klin. Woch., 35:966, 1922.

11. Imperatori, C. J.: Complete Atresia of the Esophagus. Trans. Amer. Bronchoscopic Soc., p. 74, 1931.

12. Reid, M. R.: To be published in the Trans. of the Amer. Surg. Assn. for 1932.

XCVI.

PERFORATION OF THE BRONCHUS WITH NOTES ON BRONCHOSCOPIC ANATOMY.*

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New York.

Perforation of a bronchus by anthracotic or caseous lymph nodes, carcinoma, foreign bodies, traction diverticulum of the esophagus, aneurysm of the aorta and actinomycosis is occasionally encountered, though not common. Perforation of the bronchus following instrumentation has no doubt occurred but there are no reports of such cases.

The following experience is recorded. E. B., male, age 23, was bronchoscoped by me August 20th of last year. Coincidental with rheumatic fever, seven years before, he developed a mitral and aortic insufficiency which made him a chronic invalid. He had swallowed a piece of chicken bone twenty months before admission to the hospital in October, 1929. He had no trouble of consequence except that he coughed several times a day. Four months after the accident his sputum became definitely foul and purulent. The cough and sputum increased and the patient thinks that he had fever at times. He felt sick and lost weight. When his expectoration was more profuse he felt better. He gradually lost his strength and neither the quantity of sputum nor the intensity of the foul odor were diminished. He was losing weight and several scheduled bronchoscopies had to be deferred because of his apprehension and poor condition. His cardiac disease was not considered a contraindication to bronchoscopy. It was decided that it would be best to perform bronchoscopy under tri-brom-methanol anesthesia to eliminate apprehension and at the same time to eliminate the use of cocain.

He had been suffering with shortness of breath for six months before admission to the hospital. X-ray study, eleven months after

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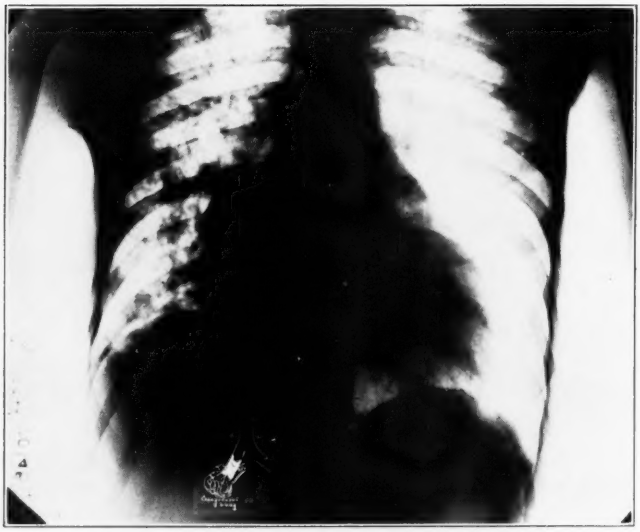

Fig. 1. Roentgenogram taken a few days before bronchoscopy.

the inhalation of the foreign body, revealed obstruction to the passage of air into and out of the right lower lobe of the lung. (Fig. 1.) During the past two months he had coughed throughout the day and night, and the expectoration amounted to more than twelve ounces a day. (Fig. 1.)

Bronchoscopy was performed by means of a 9 mm. Yankauer tube. A large amount of purulent exudate was aspirated from the lower lobe bronchus area. The lower lobe bronchus was found occluded by firm granulation tissue. This was removed with forceps and with it a small piece of bone. It appeared to be a piece of beef bone, about 8 by 3 mm. About five forceps' jaws of granulation tissue had been removed when what appeared to be a flat bony surface came into view near the mesial wall. Bronchoscopy had lasted about eighteen minutes. I grasped what appeared to be the foreign body and then released the forceps because removal met with so much resistance. With the withdrawal of the forceps there was a gush of blood through the mouth of the broncho-

scope. This continued for about two minutes, then appeared to subside. One and one-half yards of one-quarter inch gauze strip was packed into the bronchus and into what was thought to be a bronchiectatic cavity beyond. The pulse became more rapid and weaker until the patient died within a few minutes, despite all measures directed at trying to keep him alive. My impression at the time was that I had torn a large blood vessel in a bronchiectatic cavity. Autopsy was performed. The findings were as follows:

There was a break through the mesial wall of the bronchus. The right pulmonary vein was torn and the packing was found inserted through this vessel into the left auricle of the heart. A foreign body (Fig. 2) was found firmly wedged in the right lower lobe bronchus. This consisted of a chicken's vertebra, cone shaped, with its base downward. The right lower lobe was the seat of a diffuse bronchiectasis and there was gangrenous cavitation of the bronchiectatic walls. Chronic cardiovascular disease with mitral and aortic insufficiency and dilatation and hypertrophy of the left ventricle were present. Histologic sections showed acute and chronic purulent bronchitis with squamous cell metaplasia and inflammation of the branches of the lower lobe bronchus. It would appear that the bronchial wall could not withstand the pressure of my forceps and that the pulmonary vein was in very close proximity with the mesial bronchial wall because of the pulmonary congestion due to the mitral insufficiency. (Fig. 3.)

Fig. 2. Foreign body found in bronchus at postmortem examination.

As a result of the above experience, I was anxious to know whether such a mishap might have been avoided. I was also anxious to know more than I did about the anatomic relationship of the heart and great vessels to the tracheobronchial tree as they

Fig. 3. Autopsy specimen showing packing which was inserted through the bronchus and right pulmonary vein into the left auricle.

concerned the bronchoscopist in his daily work. I therefore utilized some of the material available in the autopsy room at the Kings County Hospital and dissected the tracheobronchial tree and pulmonary vessels in eight complete specimens of the thoracic contents.

There are two reasons why an understanding of the relative positions of the structures adjacent to the trachea and bronchi is of importance. First, the bronchoscopist will know where he may or may not penetrate with instruments, and second, he will be able to diagnose pathologic conditions by virtue of his knowledge of the location of the structures from which such conditions might originate.

The trachea is in relation to the arch of the aorta in its lower portion. Here the anterior tracheal wall is directly behind the aorta, which bends downward and slightly backward to cross behind the posterior wall of the left main bronchus as the vessel descends to take its place alongside the vertebral column. It is

thought by some that the left main bronchus is longer because provision must be made for the support of the arch of the aorta.

The heart and great vessels fill the space between the main bronchi so that there is no space remaining for pulmonary tissue. It would appear that the position and course of each vessel determines to some extent the position and length of the bronchi.

The main bronchi in their extrapulmonary portion do not have cartilage in their complete circumference. As soon, however, as the bronchus enters the pulmonary tissue the entire circumference has cartilaginous structure. This is important to the bronchoscopist, for it enables him to report that the lesion which he encounters is in the mediastinal or pulmonary portion of the bronchus.

The pulmonary artery lies just below and slightly anterior to the tracheal bifurcation and is separated from the left main bronchus by lymph glands and areolar tissue. The pulmonary artery bifurcates slightly to the left of the bifurcation of the trachea. It occupies the midline, while the bifurcation of the trachea is slightly to the right.

The right pulmonary artery covers the right main bronchus at the bifurcation. The left pulmonary artery crosses in front of the left main bronchus lying below it at the bifurcation and gradually coursing diagonally upward to the point of its division into the branching of its two main lobar branches. Each artery begins more or less horizontally and is in relation with the anterior wall of the main bronchus, and spreads fanlike to send branches to the main bronchial divisions. On the right side the branch to the lower lobe goes behind the middle lobe bronchus, while on the left side the lower lobe branch descends behind the upper lobe bronchus. The artery has a constant distribution to the bronchi and their branches. The arterial distribution duplicates the bronchial tree. The artery is always near the cartilaginous portion, never near the membranous portion of the extrapulmonary bronchi.

There are four pulmonary veins, two for each lung. A single trunk collects the blood from each lobe, and a vessel accompanies each bronchial element. The pulmonary veins appear to leave the

bronchi somewhat and are not as close to these structures as the arteries.

Ewart quotes Quain as follows: "The root of the right lung lies behind the superior vena cava and part of the right auricle, and below the vena azygos, which arches over it to enter the superior vena cava. That of the left lung passes below the arch of the aorta and in front of the descending aorta. The bronchi, together with the bronchial arteries and veins, the lymph vessels and the lymph glands, are placed in a plane posterior to the great vessels, whilst the pulmonary veins are in front of the arteries.

"On the right side the undivided portion of the bronchus is altogether above the pulmonary artery; on the left side the undivided portion of the bronchus, which is considerably longer than on the right side, extends to below the level of the left pulmonary artery which crosses it. On both sides the pulmonary veins are below the corresponding arteries."

The veins send branches to accompany the arteries and in addition send numerous branches to the lung parenchyma. The arteries and veins have a constant relation to the bronchi. In the specimens examined the artery was always found above or lateral to the bronchus and the vein was always found below or mesial to the bronchus. The veins have a single trunk for each lobe and there is a single vessel for each lobule.

Miller states that the relations of the three structures are as follows: "The pulmonary artery follows in all its subdivisions the subdivisions of the bronchial tree. As each main branch of the pulmonary artery arches over its corresponding stem bronchus it comes to occupy a position posterior and slightly lateral to the bronchus. The pulmonary vein's main trunks are situated anterior and mesial to the stem bronchi. The veins are never as closely associated as the artery."

SUMMARY.

A case is recorded in which the bronchus was perforated and the right pulmonary vein torn during an attempt to remove an impacted foreign body of long standing. Packing which was directed to stop the bleeding was introduced through the pul-

monary vein into the left auricle, where it was found at autopsy. The patient was suffering from severe chronic cardiac disease which influenced the proximity of the pulmonary vein, which was no doubt dilated. The bronchial walls were found to be the site of chronic and acute purulent inflammation. A brief discussion of the anatomic relationships is given.

12 E. 86TH ST.

BIBLIOGRAPHY.

Miller, Wm. Snow: Stereoroentgenograms of the Injected Lung as an Aid to the Study of Lung Architecture. Am. Rev. Tuberculosis, Vol. 2, 11, 659-62, Jan., 1919.

Ewart, William: The Bronchi and Pulmonary Blood Vessels. London, J. A., Churchill, 1889.

Waters, A. T. H.: The Anatomy of the Human Lung. J. Churchill, Liverpool, 1860.

XCVII.

BRONCHOSCOPIC LOCALIZATION OF LUNG ABSCESS.

RUDOLPH KRAMER, M. D., AND AMEIL GLASS, M. D.,

NEW YORK.

The purpose of this paper is the presentation of a method for localizing lung abscess. This method defines the position of the abscess in the lung and in its relation to the overlying thoracic cage. It is of practical importance in that the bronchoscopist can help answer the question of the thoracic surgeon, "Where shall I make my operative approach?" This question must be answered if the surgeon wishes to avoid failure in finding the abscess and to prevent serious and often fatal postoperative complications, such as infection of the free pleura, by entering the chest at the wrong place, and hemorrhage and severe suppurative or gangrenous pulmonary infections by traversing healthy lung tissues in order to reach the abscess. One must bear in mind that the correct surgical approach to a lung abscess enables the operator to enter the lung through adherent pleura and to find the abscess cavity close to the surface of the lung.

Furthermore, this method affords information as to whether one or more portions of one lobe or whether several lobes are involved, and thus it aids in evaluating the prognosis and type of therapy to be employed. While the cases selected by us as illustrations of some of the problems in lung abscess have all been treated surgically, this does not mean we are committed to any rigid form of procedure. The thoracic group at Mt. Sinai Hospital is at present directing its attention toward the surgical treatment, and it is for that reason that our material has been drawn from cases treated by surgery. In addition, it is important, in the bronchoscopic treatment of lung abscess in that the bronchoscopist, having determined which branch or branches were involved originally, knows toward which areas to direct treatment subse-

quently and can follow the progress of the disease more closely. The adoption of a definite nomenclature for the various accessible branches which are bronchoscopically important should make bronchoscopic reports understandable not only to other bronchoscopists but also to thoracic surgeons and internists.

This work is based on bronchoscopic, operative and postmortem findings correlated with the anatomic studies of Dr. Ameil Glass.

THE BRONCHOPULMONARY SEGMENTS AND THEIR THORACIC WALL TOPOGRAPHY (GLASS).

One of the requirements of present thoracic surgery, especially that of lung abscess, is greater accuracy in localization, and whereas in previous times localization within lobes was sufficient, at present the demands are for a more circumscribed unit. This work represents an attempt to establish a smaller and more accurate unit of localization than the lobe.

This unit, the bronchopulmonary segment, is a subdivision of a pulmonary lobe and represents not only an anatomic but also a pathologic unit. Each segment occupies a definite constant position in the pulmonary architecture and thoracic cavity, and is supplied by a constantly placed bronchus whose orifice is situated in a large lobar bronchus and easily visible to the bronchoscopist. The identification of the orifice leads to the recognition of the associated segment and knowledge concerning its size, shape and topographic position on the chest wall. The bronchopulmonary segment is therefore the anatomic basis for bronchoscopic localization, and upon its knowledge is dependent the degree of proficiency with which the bronchoscope can be used in localization.

To demonstrate and establish the form and position of each segment, the following procedures were employed:

First, the lobar bronchial divisions were studied and their constancy determined, and second, the segments were delineated on the pulmonary surfaces by the injection of fluid dyes into these bronchial divisions.

Eighty pairs of human lungs were especially prepared and dissected and the anatomy of the larger bronchi studied. The following illustrations show the divisions as they are found in 85

per cent of the lungs. In the remaining 15 per cent there are variations of these divisions in place of origin and number, which will be noted at another time, but which it may now be stated do not affect the conception of the bronchopulmonary segment.

The delineation of the segments was done in the following manner:

A fresh human lung was kept inflated to normal expansion by a constant stream of air. Beginning at the hilum, the parenchyma was then bluntly separated from all the bronchi to the fifth and sixth order with very little injury to the alveoli. This left a human lung expanded to normal shape and size with the bronchi easily accessible for injection purposes. Then into a chosen bronchus was injected by means of a needle and syringe a colored fluid dye. This spread rapidly throughout the small branches to the alveoli and colored the entire segment of parenchyma aerated by the injected bronchus. The main divisions of each lobar bronchus, as represented by the illustration (Fig. 1), were injected in this manner. Each division with its branches and associated alveolar tissue makes up the bronchopulmonary segment. It will be noted that the orifice of each bronchus is an opening placed in the main pathway of currents of air and infective materials.

When all the bronchi were injected with different colored fluids the lung was subdivided into segments, and each segment of lung acquired the color of the fluid injected into the corresponding bronchus. This procedure was carried out in forty pairs of human lungs, and the bronchopulmonary segments were found to be practically constant in form, size and position. The illustrations show the segments as they are outlined on the surface of the lung and their relationship to the thoracic cage, which was determined from studies of bronchograms in the living. The segments are given a very simple nomenclature, based upon their position in the lung. The right upper lobe consists of an apical, an anterior, an axillary and a paravertebral segment. The middle lobe, right side, consists of an anterior and an axillary segment. Both lower lobes are similarly divided into an apical, a paravertebral, an anterolateral, a posterolateral and a mesial segment. The left upper lobe is composed of those segments noted in the right

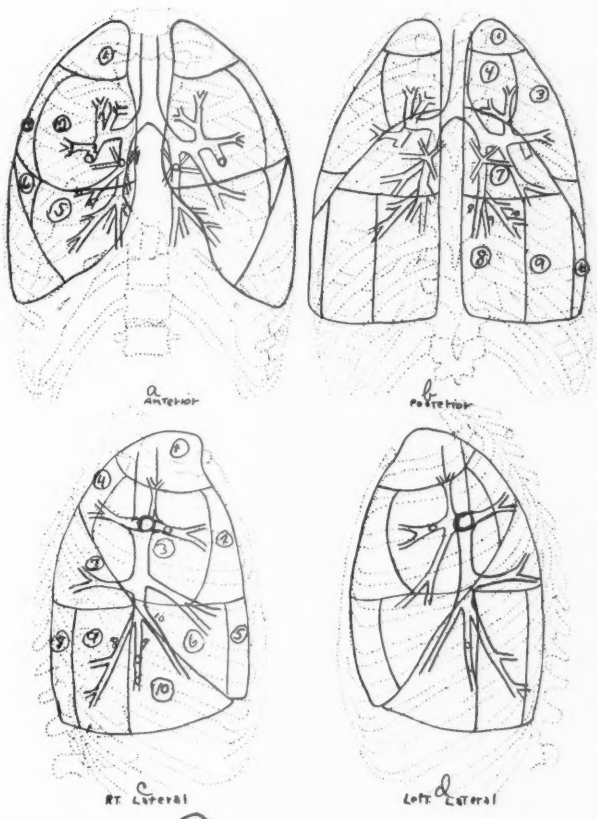

Fig. 1. Main divisions of bronchial tree with associated segments and their relationship to thoracic wall nomenclature of bronchi and segments similar.

Right upper lobe: (1) apical, (2) anterior, (3) axillary, (4) paravertebral.

Right middle lobe: (5) anterior, (6) axillary.

Right lower lobe: (7) apical, (8) paravertebral, (9) posterolateral, (10) anterolateral, (11) mesial.

upper lobe and in addition those corresponding to the right middle lobe, which on the left side are a part of the upper lobe.

Of surgical importance is the fact that each segment, except the mesial, has a surface in contact with the parietal wall and therefore is readily accessible for operative purposes. All the lower lobe segments, except the apical, have also a small representation on the diaphragmatic pulmonary surface. The mesial segment on each side lies on the diaphragm next the heart and does not come in contact with the thoracic wall. For the sake of uniformity and simplicity, the bronchi associated with these segments are given the same nomenclature.

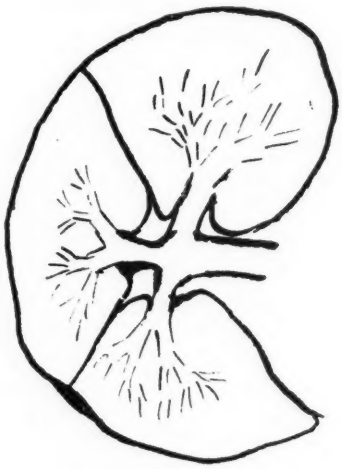

Fig. 2.

Each bronchopulmonary segment is composed of a framework of small bronchi whose trunk is an offshoot of a lobar bronchus. The trunk and branches have a definite constant direction peculiar to each segment. Accompanying these bronchi are the corresponding blood and lymph vessels and nerves. The direction of the bronchi and vessels necessarily determines the location of infections borne by these structures. The segment is an area for potential infection with an inlet from a large lobar bronchus. The semidiagrammatic representation of a cross section of the upper lobe illustrates this point. (Fig. 2.)

A study of pathologic material reveals the fact that practically all putrid abscesses have a definite position in one of these broncho-pulmonary segments. The abscess generally involves most of the segment and always approaches most closely or touches that surface of the segment towards which its main bronchus points. Illustration. (Fig. 3.)

The explanation of such anatomic location of this disease is as follows:

A segment is really the area of distribution of one of the larger bronchi and arteries. It has a bronchial orifice open wide in the path of infective material. When this enters such an orifice in

Fig. 3.

large enough quantities to cause an abscess it is distributed through and generally limited to a segment.

The significance of these facts is obvious. The broschoscopist holds the key position for information concerning suppurative pulmonary disease, for in the visualization of the bronchial orifice is revealed the position and extent of the pathologic process in the lung and its relationship to the thoracic cage. Also extension of the disease in the periphery of the lung into the domain of another segment is very early recorded at the corresponding orifice.

CLINICAL APPLICATION (KRAMER).

The description and nomenclature of the bronchi of interest to us has been given you by Dr. Glass. Although the more distal branches of some of these bronchi can be visualized readily, they can, as a rule, be disregarded for our purposes. On the other hand, some of the third order bronchi cannot be visualized at times. This is practically always limited to the upper lobe branches, particularly the apical and paravertebral. I have found that failure may be due to the following conditions: the patient's neck is short and thick; the lung is fixed in its upper portion; there is pressure on the upper wall of the upper lobe bronchus from above or on the lateral wall of the main bronchus so that a bulge of the main and upper lobe bronchial walls occur. In some cases pneumothorax has made feasible bronchoscopic exploration of bronchi previously inaccessible. But much as one might be tempted to avail oneself of this procedure, we believe it should not be used in lung abscess, because it is often followed by disastrous consequences. If repetition of bronchoscopy does not succeed in yielding us the desired information, it is better to desist from further efforts and depend on information obtainable from roentgen rays, bronchography, etc.

In addition to the usual bronchial distribution, we have encountered, in a few cases, anomalous branches which were infected. These were generally branches arising from the right tracheal wall, one-half to one centimeter above the carina and directed downwards and to the right to supply a portion of the apex of the right upper lobe.

The bonchoscopic examination is made to determine which bronchus or bronchi are draining an abscess. After the main and branch bronchi have been cleared of pus by aspiration, the reappearance of pus is watched for in each branch. Each branch in which pus reappears after aspiration is a bronchus draining infected lung. Occasionally, in the very early stages of acute lung abscess, pus may not be found on bronchoscopy. This may be due to the fact that there is a temporary blocking of the lumen of the bronchus by slough, thick secretion or swelling of the bronchial mucosa. Repetition of the bronchoscopy in a few days will usually prove successful in locating the abscess.

The most frequently involved branches have been, in our experience, the apical branch of the lower lobe, the anterior and the paravertebral branches of the upper lobe. The middle lobe branches and the axillary branches of the upper lobe have been less frequently involved. The remaining bronchi are involved relatively seldom.

I should like to cite briefly a few cases illustrating some of the problems arising in the diagnosis and treatment of lung abscess with relation to the localization.

Case 1.—Acute lung abscess; three weeks' duration. X-ray showed infiltration right lower lobe with cavity. Bronchoscopy showed foul pus from the apical branch of the right lower lobe. Thoracotomy showed abscess under adherent pleura on posterior mediastinal aspect right lower lobe; sixth rib resected posteriorly.

Case 2.—Subacute lung abscess, two and one-half months' duration. X-ray showed shadow root of the left lung with irregular cavity. Bronchoscope showed creamy pus apical branch left lower lobe. Thoracotomy showed cavity here; abscess drained and patient well in short time.

Case 3.—Subacute abscess, four months' duration. Patient was six years old, and presented a puzzling picture. He had nonproductive cough, slight fever for a short time, clubbing of the fingers, signs of infiltration left upper lobe. X-ray showed dense infiltration upper half of left chest with a cavity opposite the third rib anteriorly. Bronchoscopy showed foul pus coming from the apical branch of the left lower lobe. After bronchoscopy foul odor of patient's breath and pus was noted for the first time. Posterior thoracotomy; resection of sixth rib showed an excavation of upper part of lower lobe. Physical examination and X-ray erroneous in this case. Bronchoscopy made the diagnosis and proper surgical approach possible.

Case 4.—Chronic abscess, six months' duration. Slight fever, only at onset; foul sputum; later symptoms of pyopneumothorax. X-ray showed right lower lobe consolidation with a cavity. Bronchoscopy showed pus from apical branch right lower lobe. Posterior thoracotomy showed empyema with a piece of thread in the pleural cavity. Abscess in apical portion right lower lobe drained. Patient was a seamstress and had apparently aspirated a piece

of thread, following which she had a lung abscess which ruptured and formed localized pyopneumothorax.

Case 5.—Chronic abscess; onset one year before admission. Symptoms cleared up in one week and well for one year; then had fever, pain in chest, cough and foul expectoration. Physical signs and X-ray showed lesion in upper posterior part of left lower lobe, with cavity. Bronchoscopy showed foul pus from apical branch left lower lobe. Posterior thoracotomy; abscess drained.

Case 6.—Chronic abscess, fever, cough, foul sputum. Physical signs and X-ray showed lesion with cavity in right upper lobe anteriorly. Bronchoscopy showed foul pus paravertebral branch right upper lobe. Because of physical signs and X-ray, an anterior thoracotomy over the right upper lobe was done; abscess opened at depth of 8 centimeters; profuse hemorrhage. Patient did poorly and was subjected to several operative procedures in further attempts to relieve her. Finally a posterior thoracotomy over the right upper lobe was performed and a superficially lying abscess drained and patient got well. As a result of an incorrect operative approach the cavity was reached only after traversing a good deal of healthy lung; profuse hemorrhage resulted and patient failed to get well until the primary abscess (as revealed by bronchoscopy) was drained properly.

Case 7.—Chronic abscess. Physical signs, right lung posteriorly. X-ray showed mid two-thirds of lung involved. Bronchoscopy showed foul pus from paravertebral branch right upper lobe and from apical branch right lower lobe. Posterior thoracotomy done, but patient died ten days later because of gangrenous extension and spillover. Postmortem showed the cavities in posterior portions of right upper lobe and right lower lobe as indicated by bronchoscopy and X-ray.

Case 8.—Chronic abscess. Signs in chest equivocal; X-ray showed lesion with a cavity in right upper lobe. Bronchoscopy showed pus coming from the paravertebral branch of the right upper lobe and apical branch right lower lobe. Thoracotomy done and an anterior right upper lobe approach done with negative findings. One week later posterior thoracotomy done, seventh rib, and a bilocular cavity found. Patient died of pneumonia. Postmortem showed pneumonia all lobes (spillover). Abscess

in posterior part right lower lobe had been drained; an abscess above this in the posterior lower part of right upper lobe had not been opened. This case was treated several years ago, and the information obtained by bronchoscopy was neglected. As a result the operative approach was too low and the upper cavity was not recognized and drained, with the result that a spillover infection occurred.

Case 9.—Chronic abscess. Numerous chest operations had been done at various hospitals on this patient. Then three abscesses were opened, one in middle lobe, one in right upper lobe anteriorly, and one in posterior lateral portion right lower lobe. One year later patient was still bringing up foul pus and ran septic temperature. Physical signs and X-ray gave no clue as to the focus, because of the scarring and infiltration of the lung. Bronchoscopy showed foul pus from axillary branch, which arose from anterior division of right upper lobe. Thoracotomy fourth rib, mid-axilla, abscess opened; tract followed from this to fistula in anterior chest; patient ceased from what appeared to be a cerebral air embolus. Postmortem showed that all cavities had been drained by operation; the last cavity having been indicated only by bronchoscopy.

Case 10.—Chronic abscess; had four operations with relief but still expectorated foul pus. X-ray showed infiltration right upper lobe; no cavity. Bronchoscopy showed pus from anterior division right upper lobe. Anterior thoracotomy right upper lobe, lower portion, revealed smooth lined abscess cavity with foul pus. Patient well.

Case 11.—Chronic abscess. Patient admitted with picture of ruptured lung abscess. History of cough, pain in left chest, septic temperature, hemoptysis. Empyema and abscess of left lower lower lobe drained. One month later second encapsulated pyopneumothorax opened. Symptoms continued. Three months later enormous abscess of left lower lobe drained. Three months later child was still febrile and coughing. X-ray showed dense homogeneous shadow lower half of left chest with less infiltration above; no cavity. Bronchoscopy showed pus from paravertebral branch left upper lobe and a small amount from the anterior division of the left upper lobe. Thoracotomy showed an abscess

in lower part of posterior portion of left upper lobe, which communicated in series with three other abscesses along and above the interlobar fissure. At the axillary region they crossed the fissure to the upper part of the left lower lobe and finally communicated with the pyopneumothorax cavity. The lesion apparently started in the upper lobe and there were successive spillover infections in other bronchi with involvement, finally, of the lower lobe and rupture into the pleural cavity. Surgery began with the latest lesion and worked back to the primary lesion. Bronchoscopy afforded the only definite information as to the existence and location of the residual abscess.

Case 12.—Acute lung abscess, three weeks' duration. Pain in right lower lobe, fever, cough, foul sputum, hemoptysis. Physical signs and X-ray showed infiltration over right lower lobe. Bronchoscopy negative except for foul odor. Patient became worse. Thoracotomy done (eighth rib posteriorly); adherent pleura found but no pus. Specimen of tissue removed showed necrotic tissue. Patient thought to have lung tumor; died in five months. Postmortem showed lung abscess in right lower lobe. Bronchoscopy done in the early stages of this lesion failed to reveal source of pus. We have since learned to repeat the bronchoscopy after a few days.

These cases are presented as illustrations of some of the problems arising in the treatment of lung abscess and of the aid which the bronchoscopist can afford in the solution of some of these difficulties. Intelligent treatment of lung suppuration and progress in the elucidation of the etiologic, pathologic, diagnostic and therapeutic phases of lung abscess can only come about by the close co-operation of a group of men who, although they are specialists in their own fields, are thoroughly conversant with the problems and viewpoints of each other. Dr. Sidney Yankauer started the work of localizing lung abscess by bronchoscopy a number of years ago. When the necessity for accurate localization became more apparent, after the formation of a group for the study of thoracic diseases, it was possible to investigate the problem in its various phases, because of the co-operation of a number of men, each bringing to bear his special knowledge and each trying to assimilate the work and the viewpoints of the other members of the group.

XCVIII.

BRONCHOSCOPIC RELIEF OF POSTOPERATIVE PULMONARY COMPLICATIONS.*

ETHAN F. BUTLER, M. D.,

ELMIRA, N. Y.

This discussion is definitely limited to a consideration of two major points: First, the varied etiologic factors responsible for post-operative pulmonary complications; second, the rôle of early bronchoscopy in preventing, aborting or relieving some of these complications. No effort will be made to fully describe the final pathology that may be encountered nor will there be consideration of the bronchoscopic treatment of established pulmonary lesions long after the period of immediate surgical convalescence.

The etiology of postoperative pulmonary complications has long been a matter of interest. Many theories have been advanced, and no one theory has been sufficient to explain all cases. It is necessary to consider all factors which may play a primary rôle in the causation of these complications, and some additional factors which may play very important secondary rôles.

Etiologic factors of primary importance are:

1. Embolism.
2. Pulmonary congestion.
3. Infection by lymphatics or direct extension.
4. Foreign material in the airways.
5. Disturbances of the intrinsic pulmonary neuromuscular mechanism.
6. Disturbances of the extrinsic respiratory mechanism.

Factors of secondary importance are:

1. Pre-existing pulmonary disease.
2. Infecting organisms which may accompany emboli, foreign material in the airways or be resident in the lower airways.
3. Irritation from anesthetics.
4. Lowered resistance on the part of the patient.
5. Infection in the upper air passages or mouth.

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Cutler[1] [2] and his associates stressed the importance of embolism and came to the conclusion that it was the principal factor. That conclusion has not been unanimously upheld, although the importance of embolism is generally admitted. The pulmonary circulation receives all blood from the right heart. Any particulate matter which is too coarse to pass the pulmonary capillaries or which may be trapped in any portion of the narrowing stream bed will result in pulmonary infarction or embolism. The end pathology may be sterile infarcts, pneumonitis, lung abscess, gangrene and sudden death from massive embolism. In his original contributions Cutler included embolism by way of the lymphatics. In this classification the rôle of the lymphatics is considered under another heading.

Myocardial failure, certain valvular diseases of the heart and other vasomotor disturbances may result in stasis of pulmonary circulation, pulmonary edema, pleural exudate, so-called "hypostatic pneumonia" and other conditions. The outcome is dependent upon control of the circulatory disturbance.

Infecting organisms are placed in a group by themselves. It is true that they may accompany emboli, but there their rôle is secondary. It is true that they may be resident in lung tissue damaged by circulatory failure or involved in atelectatic changes or handicaped by respiratory embarrassment, but again their rôle is secondary. It is true that they may accompany aspirated material into the lower air passages, and again they play a secondary rôle. Under this heading is considered the extension of infection from other parts of the body by lymphatic channels, by thoracic duct or by direct extension from neighboring structures. Whipple[3] has been of the opinion that pneumonitis forms the basis of the majority of lung complications. Lobar pneumonia, bronchopneumonia, pneumonitis, lung abscess, bronchiectasis, mediastinal abscess, lung gangrene and empyema may be ultimate results of infection.

Tracheal aspiration has been vigorously debated as a factor in producing lung pathology. There is no doubt in my mind that the work of Lemon[4] and many other authors has conclusively proved its active rôle. Tracheal aspiration may result in the presence of obstructing or nonobstructing material in the tracheo-

bronchial tree. It may result in the presence of virulent pathologic organisms or organisms of low virulence or mere saprophytes in the lower air passages. It may occur during the narcosis of general anesthesia, follow vomiting during recovery from anesthesia or take place through the cocainized pharynx. The late results may be atelectasis, bronchitis, bronchopneumonia, lung abscess, lung gangrene and asphyxia.

The intrinsic pulmonary neuro-muscular-secretory mechanism and its reflex relationships with other organs should be a matter of primary importance, not only to the internist interested in the varied manifestations of allergy, but also to the abdominal surgeon and to the bronchoscopist. Reflex disturbance of this intrinsic mechanism may eventually be proved to be the principal cause of postoperative atelectasis, which, at the present time, is regarded by many investigators as being the most important factor in the production of postoperative pulmonary complications. According to the prevailing opinion, atelectasis is due to the occlusion of a branch bronchus by a plug of thick tenacious mucus, and there is a wealth of experimental evidence to prove that occlusion of a branch bronchus will be followed by atelectatic changes. However, it is impossible to disregard the opinions of Pottenger[5] and Matas,[6] and the experimental evidence of Luisada,[7] which all unite to strengthen the belief that the atelectasis may also be the result of a viscero-visceral reflex, probably vagal or parasympathetic, and that the mucus plug, when present, is also a secondary result of reflex disurbance affecting the character and quantity of the bronchial secretions. Therefore, in this discussion, atelectasis has not been mentioned by name among the primary etiologic factors, but has been grouped under these disturbances of the intrinsic pulmonary neuro-muscular-secretory mechanism and other primary factors. Atelectasis may spontaneously disappear; it may be followed by bronchitis, pneumonitis or bronchopneumonia.

The extrinsic respiratory mechanism, especially the diaphragm, is always disturbed after upper abdominal operations, and to lesser degrees and less constantly after operations on the lower abdomen and elsewhere in the body. Muller, Overholt and Pendergrass[8] have very thoroughly discussed this matter and demon-

strated the hypoventilation that results therefrom. There is reduction of vital capacity, accumulation of bronchial secretions, loss of effective cough. Bronchitis, bronchopneumonia and lobular atelectasis from occlusion may occur. If there are abundant bronchial secretions the patients may be intensely uncomfortable.

Of the secondary factors, only one will be commented upon. Pre-existing pulmonary disease, if it be of a suppurative nature, may produce flooding of the airways with pus, with resultant dissemination of disease, or even threatened or actual asphyxia. This constitutes a definite preoperative menace.

Bronchoscopy can render very definite help in appropriate cases, but this aid is necessarily limited to the removal of foreign material, the removal of excessive secretions, the reopening of occluded airways, the introduction of certain medicinal substances and the mechanical stimulation of the intrinsic neuromuscular mechanism of the lung. To what degree the passage of a bronchoscope may affect bronchial reflexes has, apparently never been determined. The effect of bronchoscopy in certain cases of asthma and the effect in reported cases of atelectasis, in which no mucus plugs were found, strongly suggests, however, that some pulmonary and bronchial reflexes may be inhibited or otherwise modified by mechanical stimulation.

In so far as embolism, circulatory failure and infection by lymphatics or direct extension are concerned, early bronchoscopy can do nothing to modify the course of the pathologic process. It may be useful in the late results, but it cannot prevent their occurrence. In so far as foreign bodies in the air passages are concerned, early bronchoscopy is, naturally, the procedure of choice and should be done without delay. In the event of flooding of the lower air passages with excessive secretions from suppurative foci in the lungs, so that asphyxia is threatened, bronchoscopic or laryngoscopic aspiration of the secretions becomes imperative. When there is no immediate threat of asphyxia, hyperventilation by inhalation of carbon dioxid and oxygen may enable the patient to cough up the purulent secretions.

In the event of nonobstructing foreign material due to tracheal aspiration of pharyngeal secretions or contents, or in the event of disturbances of the respiratory mechanism following abdominal

operation, and consequent accumulation of bronchial secretions, the first thought should be to restore and maintain the cough reflex; the second thought should be of forced hyperventilation of the lungs by inhalation of carbon dioxid and oxygen. If these measures fail to accomplish satisfactory results, bronchoscopy will give the desired result and will prevent later complications. Furthermore, patients recovering from abdominal operations are, as a class, incapable of effective cough effort. If there be any great accumulation of bronchial secretions, these patients are constantly harassed by the conflicting desire to cough and the fear of pain produced thereby. The relief afforded to these patients by thorough bronchoscopic removal of the irritating secretions is very great.

In the presence of disturbances of the intrinsic pulmonary neuro-muscular-secretory mechanism resulting in atelectasis, the first thought should be the simpler procedures: atropin as a vagal inhibitor, external mechanical stimulation by change of position, etc., and forced hyperventilation by carbon dioxid. Unless decided relief is obtained from the simple measures within twenty-four hours' time, bronchoscopy is indicated.

An effort has been made to enumerate all logical alternative measures to bronchoscopy. The most potent is forced hyperventilation by carbon dioxid, which has not only won a definite place for itself in the technic of inhalation anesthesia but which also has a definite place in the routine of after-care of surgical patients. As a variation to bronchoscopic aspiration of retained secretions there must also be mentioned the laryngoscopic pertracheal aspiration of bronchial secretions.

The question will properly be raised that bronchoscopy is a severe ordeal for a patient recently operated upon, and that the risk of wound rupture or other unfortunate sequelæ must be great. Experience with thirteen cases forms the basis for this paper. In no case did there occur any damage to the operative wound. In all cases the precaution was taken to apply a tightly fitting abdominal binder before bronchoscopy, to give preliminary morphin by hypodermic and to immediately inspect the wound as soon as the patient was returned to bed.

In one case laryngoscopic aspiration of pus was carried out as a preoperative measure. In two cases bronchoscopy was performed the day after operation—for atelectasis in both instances. Two other cases were treated two days after operation—one for atelectasis, the other for hypoventilation and retained secretions. Three cases were treated from five to nine days after operations, all of these being in the group of hypoventilation and retained secretions. In every one of the foregoing eight cases there was a rapid relief of the chest condition, and all but one made satisfactory recoveries. One case died from pyonephrosis due to a ruptured bladder, but at a date remote from the postoperative atelectasis for which bronchoscopy had been performed. In five cases bronchoscopy was done more than two weeks after operation for complications arising at late dates. There was no mortality in this group, but the results of bronchoscopy were by no means as dramatic or satisfactory as in the earlier cases. The ages of the patients varied from 21 to 59. One operation was thoracic, one on an extremity, four were upper abdominal and seven were lower abdominal. No patients died from pulmonary complications and only one died from other causes.

SUMMARY.

Primary and secondary etiologic factors responsible for postoperative pulmonary complications are enumerated.

The opinion is ventured that atelectasis is the result of a parasympathetic viscero-visceral reflex.

The opinion is ventured that the mere passage of a bronchoscope gives rise to mechanical stimulation which can modify or inhibit pulmonary reflexes.

Early bronchoscopy cannot abort or relieve postoperative pulmonary complications due primarily to embolism, cardiac failure or infection by direct channels.

Early bronchoscopy may be of great value in aborting or relieving complications due primarily to tracheal aspiration, pulmonary reflexes, hypoventilation or flooding of lungs by purulent secretions.

Alternatives to bronchoscopy, in appropriate cases, are: atropin, external stimulation, inhalations of carbon dioxid and laryngoscopic aspiration.

Operative wounds of one day to three weeks' standing have suffered no ill effects as a result of bronchoscopic treatment.

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REFERENCES.

1. Cutler, Elliott C., and Hunt, Alice M.: Postoperative Pulmonary Complications. Arch. Surg., I, 1920, pp. 114-157.

2. Cutler, Elliott C., and Hunt, Alice M.: Postoperative Pulmonary Complications. Arch. Int. Med., XXIX, 1922, pp. 449-481.

3. Whipple, Allen O.: A Study of Postoperative Pneumonitis. Surg., Gyn. and Obst., XXVI, 1918, pp. 29-47.

4. Lemon, Willis S.: Aspiration: Experimental Study. Arch. Surg., XII, 1926, pp. 187-211.

5. Pottenger, F. M.: Important Reflex Relationships Between the Lungs and Other Viscera. Jour. Thor. Surg., I, 1931, pp. 75-90.

6. Matas, Rudolph: On the Present Status of the Postoperative Pneumopathies. International Clinics, II, Series 41, 1931.

7. Luisada, Aldo: La Contractilité Active du Poumon etudieé au Moyen de L'electrobronchographie. Arch. Méd.-chir. de l'app. respir., V, 1930, p. 320.

8. Muller, George P., Overholt, Richard H., and Pendergrass, Eugene P.: Postoperative Pulmonary Hypoventilation. Arch. Surg., XIX, 1929, pp. 1322-1345.

XCIX.

SPONTANEOUS PERFORATION OF THE AORTA FROM AN OPEN SAFETY PIN IN THE ESOPHAGUS, WITH FATAL HEMORRHAGE, OCCURRING IN A CHILD, SEVEN MONTHS OF AGE, DURING THE PASSAGE OF THE PIN THROUGH ESOPHAGUS AND GASTRO-INTESTINAL TRACT.

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PHILADELPHIA.

G. P., aged seven months, was referred by Dr. Frank Vanderbogert with the following history: The child had been delivered normally at full term, and had received no medical attention whatever except the usual care after delivery. It was breast and bottle fed and was normally developed. Two weeks before the present illness the parents noted that the child was paler than usual and seemed to have slight difficulty in swallowing. This continued up until four days before admission when the child had a vomiting attack and seemed slightly feverish. One day before admission the child vomited a small quantity of dark blood and shortly afterward a considerable quantity, several ounces, of bright red blood. The mother then took the baby to Dr. Vanderbogert, who examined the child and immediately had an X-ray examination, which showed an open safety pin in the esophagus with the point up and toward the left, just above the diaphragm. (Fig. 1.) There was no further bleeding at that time and the child was referred by Dr. Vanderbogert for removal of the pin. On admission to the Graduate Hospital of the University of Pennsylvania, eight hours' later, fluoroscopic examination showed the pin to be in the stomach. (Fig. 2.) The child's condition was fairly good; temperature was 101. He had been given glucose and fluids by bowel, everything having been withheld by mouth after the time of the first hemorrhage.

Fig. 1. Open safety pin in the lower thoracic esophagus. Film made two hours after the first hemorrhage. Arrows outline the enlarged heart.

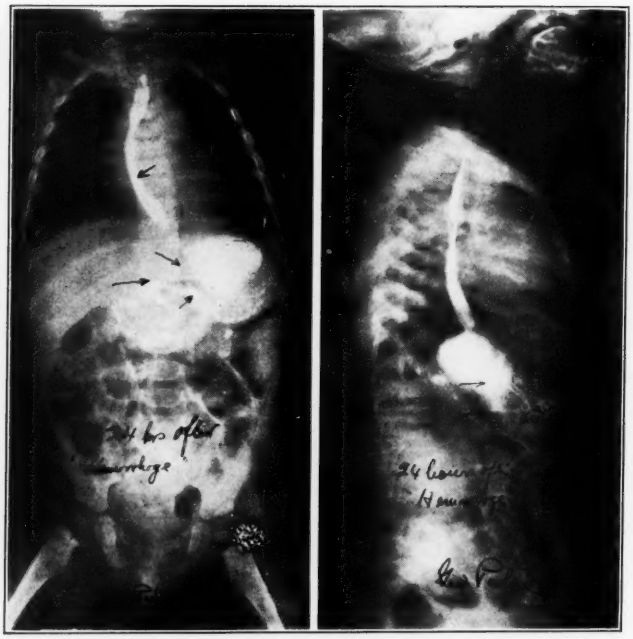


Fig. 2. Anteroposterior and lateral roentgenogram with an opaque mixture showing the open safety pin in the stomach. Made 24 hours after the first hemorrhage. Upper arrow indicates the bismuth in the esophagus being displaced by the enlarged heart.

There was no indication for attempting removal of the pin from the stomach. Glucose was given by bowel and subcutaneous. The child's general condition remained about the same, but because of the severe anemia, hemoglobin being 30 per cent, the child was typed for transfusion. The temperature during the night after admission went as high as 104. It had been noted on X-ray examination after admission that the cardiac area was enlarged generally. (Fig. 2.) With the child at rest, there had been no further bleeding until twenty-four hours after admission, when a very profuse hemorrhage occurred, with vomiting of blood and passing of blood by bowel. Another X-ray examination was

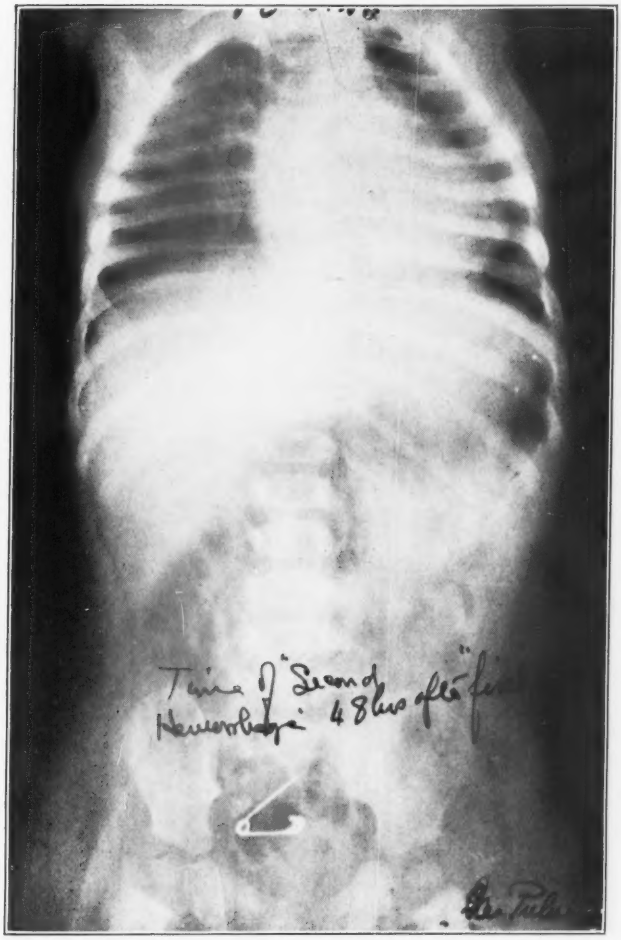


Fig. 3. Bedside roentgenogram made at time of second hemorrhage, 48 hours after the first.

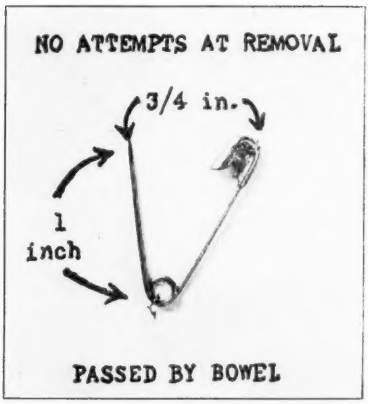


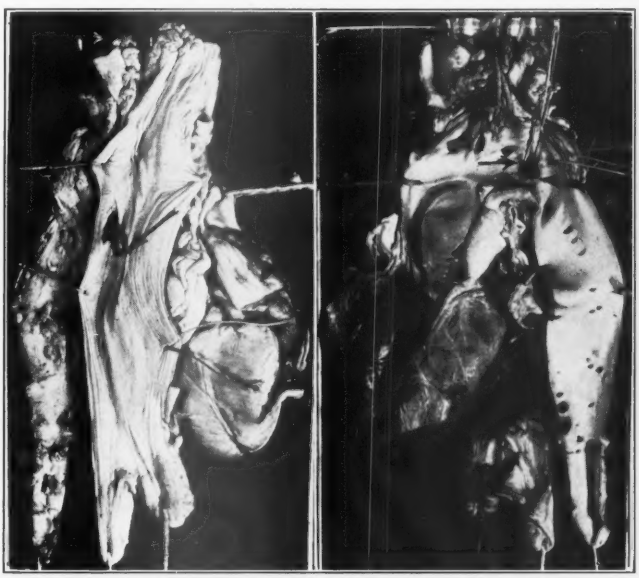
Fig. 4. Open safety pin which passed spontaneously by bowel.

made without moving the child from the bed, and the safety pin was found to have passed into the rectum. (Fig. 3.) It seemed probable that the hemorrhage was not due to trauma by the pin after it had reached the stomach. The child was transfused and immediately on the introduction of the blood in the arm bloody fluid came out of the child's mouth. The child died in the course of a few hours, the safety pin having passed from the rectum. (Fig. 4.)

Postmortem examination showed a fistula that would admit a good sized darning needle leading from the upper thoracic esophagus outward and upward toward the left into the aorta. (Fig. 5.) Dr. E. A. Case reported as follows:

"1. Exsanguination. 2. Acute purulent pericarditis. 3. Tear in the wall of the esophagus and a tear in the wall of the aorta corresponding to it. 4. Minute embolic abscesses of the kidney."

Checking up the history with the family, it was found that a safety pin had been missed about a month before. There had been apparently no indication of its having passed into the child's esophagus until a few days before the first hemorrhage, when the child developed difficulty in swallowing. Even this was not severe enough for the parents to call a doctor until the hemorrhage



A *B*

Fig. 5. Postmortem specimen of the esophagus. Photograph *A* showing perforation from the esophagus with a glass rod inserted. *B* showing the glass rod emerging from the perforation in the aorta that the pin had passed through.

occurred. At no time was there any attempt at removal of the foreign body or passage of a stomach tube or instrumentation of any sort. The perforation of the esophagus and aorta by the pin was evidently a slow process due to the continued pressure of the pin and the swallowing movements. (Fig. 6.) Severe hemorrhage did not occur until the pain had been dislodged from the perforation in the esophagus.

The point that impressed me most in this case was its utter hopelessness at the time the patient first came under the observation of a physician. Had the pin remained in the esophagus we undoubtedly would have attempted its removal. The removal of the pin, of course, would not have prevented the fatal hemorrhage and would not have relieved the empyema of the pericardium

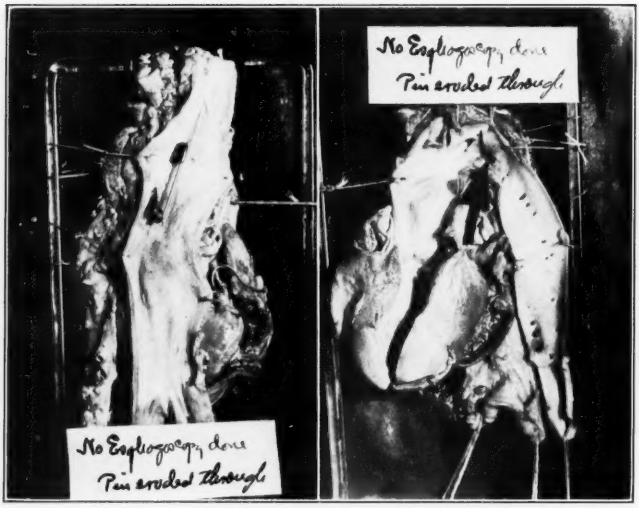


Fig. 6. Photograph of the postmortem specimen with the safety pin replaced in the esophagus with the pin perforating into the aorta. This was evidently the position of the pin before it was dislodged before the first hemorrhage occurred.

which undoubtedly followed the spontaneous perforation of the pin. This case teaches two important lessons. First, the importance of immediate X-ray examination of the esophagus in every child who has the *slighest difficulty* in swallowing. Second, the importance of teaching the prophylaxis of foreign body accidents to the laity. If the safety pin had been kept out of the reach of the child or even closed, the accident would not have happened, or if it had with a closed pin would not have been fatal.

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C.

THE MANAGEMENT OF PERFORATIONS OF THE CERVICAL ESOPHAGUS.*

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The management of perforations of the cervical esophagus places upon the endoscopist a responsibility of unusual gravity. Under few other circumstances is the accurate evaluation of symptoms and signs so important to the safety of his patient. Shall his course be a conservative one of "watchful waiting," disturbing though it may be; or frankly recognizing the rapidity with which infection may gravitate to the mediastinum, shall he elect early external drainage? His burden is not lightened by a study of the literature, for there he finds a surprising divergence of opinion among competent observers concerning the procedure of choice. Cases are reported in which a conservative policy pursued often in the presence of the most outspoken signs of perforation has proved ultimately successful, and others in which the same policy has failed under circumstances apparently far less desperate. Still other cases are described in which endoscopic drainage has proved so successful that its use is urged in preference to external drainage. Finally, another group, in summarizing their experiences, advocate early external drainage as the safest procedure whenever definite evidence of perforation exists. This divergence of opinion has made for confusion with regard to a condition in which it is particularly important that no confusion exist. It is in striking contrast to the attitude of general surgeons toward the management of serious infections in other parts of the body. The writers hope that from this paper and the discussion which it stimulates there may arise a clearer conception of the principles of management of this important complication.

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Anatomy.—Certain peculiarities in the anatomy of the cervical esophagus deserve mention. The posterior wall at the level of the cricoid cartilage contains a V-shaped gap about one inch in length where the outer layer of longitudinal muscle fibers is completely lacking. Perforation is particularly easy in this weakened area. The cervical esophagus furthermore possesses no serous coat and, according to Saint,[1] its blood supply is the poorest of all the segments. These conditions, continues Saint, make for "easy necrosis and extravasation of infectious mucus secretions."

It is of the utmost importance to our subject that the relations of the cervical esophagus to the deep fascial spaces be thoroughly understood. The superficial layer of the deep cervical fascia with its two deep processes, the pretracheal and prevertebral fascia, divides the neck into three compartments, anterior, middle (visceral) and posterior. The anterior space, between the superficial layer and the pretracheal fascia, contains the anterior belly of the omohyoid and the sternohyoid muscles. The middle or visceral compartment, bounded by the pretracheal and prevertebral fascia, contains the thyroid, trachea and esophagus. The carotid sheath forms the lateral boundary of this space. The posterior compartment contains the prevertebral and deep neck muscles. There is a wealth of clinical and experimental evidence to prove the direct communication existing between the visceral and retrovisceral spaces and the mediastinum. Solutions injected into either of these spaces can be traced directly into the chest cavity. The communication from the posterior or retrovisceral space is particularly direct. Furstenberg[2] regards it as "the most important pathway between the upper cervical region and the mediastinum." The reason for this close association lies in the fact that in the embryo the heart and mediastinal structures originate in the neck and later migrate downward to their permanent position in the chest, carrying their enveloping fascia with them. The proximity of the carotid sheath to the visceral and retrovisceral spaces must be particularly emphasized. There are many cases on record of extension of a periesophageal infection to this space. Its dense, elastic nature enables it to hold pus under great tension, and infection in this region is accordingly of the utmost gravity because of its devastating effect on the deep neck vessels.

Symptoms.—The symptoms and signs resulting from perforation of the cervical esophagus vary considerably with the position and nature of the injury. There are, however, certain general effects, which are regularly observed. Fever, pain and swelling, as pointed out by Killian,[3] in his masterful survey, form a symptom-complex of positive diagnostic value. Pain is the first symptom noted and may increase so rapidly in severity that deglutition becomes impossible. It is accompanied by marked tenderness over the site of perforation and swelling, which may be caused by emphysema, cellulitis or abscess formation. Emphysema may occur slowly, limited at first to the deep peri-esophageal tissues, or as the result of straining may be palpable almost immediately in the subcutaneous tissues. Dysypea may be observed as the result of pressure or swelling about the introitus of the larynx. Fever is an early and important index of the severity of the infection. Killian[3] regards it as the "dominating symptom." A rapidly rising fever must be viewed with grave concern. A suddenly falling temperature may indicate sudden evacuation of the infection into new spaces rather than a favorable sign. With the formation of pus, increasing general malaise, chills or delirium may occur. Evidence of extreme shock usually appears immediately following instrumental perforation.

In order to understand the symptoms and signs of perforations as well as their significance from the standpoint of treatment, it is important at the very beginning to distinguish the various types encountered.

1. In the first place, perforation may occur slowly by erosion. This not uncommonly results from sharp particles of bone or fish bones which have been lodged for several days before removal. A periesophagitis occurs with or without abscess formation. Walling off of the infection by the protective reaction of the peri-esophageal tissues commonly follows. This type of infection not infrequently drains spontaneously at the time of or soon after the removal of the foreign body. Pain, tenderness, swelling, moderate fever and even deep emphysema may occur, but the entire clinical picture is commonly subacute rather than "stormy."

2. A second type of perforation may be described as the "minute type." This is frequently encountered in cases of pins or

needles lodged in the cervical esophagus. The progress of the infection is an insidious one and several days may elapse before definite external signs manifest themselves. A small abscess slowly forms in the periesophageal tissues. This may maintain its communication with the lumen of the esophagus and evacuate itself spontaneously without much symptomatic disturbance or external signs. The perforation, on the other hand, may seal off, leaving a closed abscess which, after a period of apparently minor disturbances, may suddenly invade the contents of the carotid sheath or descend rapidly into the mediastinum. Myerson[4] reported a case of fatal septic thrombophlebitis with multiple embolic abscesses in the lungs resulting from a minute perforation which at the time of removal of the foreign body (a small safety pin) was considered perfectly harmless. Killian[3] also stresses the insidiousness of this type of perforation as well as the serious sequelæ that not infrequently complicate it.

3. As a third type may be cited perforation occurring in the posterior wall of the cervical esophagus. This permits infection immediately to enter the retrovisceral space, which Furstenberg[2] considers the most direct pathway from the cervical region to the mediastinum. The immediate clinical signs, however, may give little hint of the seriousness of the condition, because of the depth of the infection.

4. As a fourth type may be considered gross, immediate perforation, such as results from false passage during instrumentation or tearing of the wall during removal of a sharp foreign body. This is the most serious of all types and the resulting symptoms and signs are most pronounced. Severe pain, often radiating downward into the chest or shoulder, is immediately noted. Local tenderness is exquisite. Emphysema commonly develops with astonishing rapidity so that it can be palpated almost immediately. The patient within a brief period appears seriously ill and may exhibit every indication of severe shock. Fever occurs rapidly and follows a septic course. There is a high leucocytosis. The picture is stormy and the exact opposite to the subacute type already described.

Diagnosis.—The evidence gained by direct inspection through the esophagoscope is of the utmost value and may be an impor-

tant factor in determining the plan of treatment. The X-ray is also an invaluable aid. Iglauer[5] and Killian[3] independently called attention to the importance of the lateral view, which in the presence of perforation may show a marked increase in thickness of the narrow clear space between the esophagus and the vertebral column. Bubbles of air may be detected in this area long before evidence of emphysema appears in the neck, and occasionally, as in one of Iglauer's cases, the foreign body itself may be observed outside of the esophagus in the area of emphysema. The X-ray is also of invaluable assistance in following the progress of infection in the peri-esophageal tissues, as is well illustrated in certain of the cases reported. If the use of opaque media is necessary to establish the diagnosis, the nonirritating properties of lipiodol make it especially desirable. In large perforations bits of this material may be detected in the peri-esophageal tissues.

Review of Opinions Concerning Management.—As has already been indicated, the experiences of many observers have led to widely divergent views concerning the general plan of management of these cases.

Schlemmer,[6] in 1920, reviewed a series of 529 cases of esophageal foreign bodies and expressed himself strongly in favor of early external drainage, basing his principal indication on the presence of emphysema.

Killian,[3] in 1922, reviewed a series of 389 cases of which 169 were operated upon. He, too, favored external drainage. As absolute indications he cited emphysema, increasing fever, pain, tenderness and swelling. He was particularly impressed by the importance of high fever as a positive indication for surgical intervention. He also included dangerous hemorrhage and the presence of a tracheo-esophageal fistula. He emphasized the potential dangers of even the most minute perforation.

Marschik and Jurasz[7] urged "prophylactic collar mediastinotomy" and took the stand that it is safer to operate too often than to withhold operation.

King,[8] in 1929, reported a series of six cases, including four cases of perforation of the cervical esophagus. Although he rec-

ognized the difficulties of a definite decision, he favored external drainage as the safest means of controlling the situation.

Iglauer[5] has frequently placed himself on record as favoring early external drainage in order to safeguard the patient.

On the other hand, von Eicken,[9] in 1926, took the stand that there are exceptions to the dictum that immediate external drainage is indicated by the presence of emphysema and supported his view by the records of eight cases. In two of these, for example, fever accompanied by definite roentgenologic evidence of air in the prevertebral space was present. Both patients recovered after a few days of conservative treatment. Only when rising fever and a decidedly unfavorable change in the general condition intervene did he consider external drainage necessary. It is of considerable interest to note the comment on this paper by Hajek,[10] who remarked that he personally lacked the courage to follow a waiting policy under such circumstances.

Myerson,[4] in 1927, reported a series of twelve cases and concluded that "external operation may be deferred, even in the presence of severe local and systemic reaction." He described two cases of exceptional interest in this respect. Both showed high fever, emphysema, severe pain and local tenderness. In one, an instrumental perforation of the cervical esophagus was known to have occurred. Both cases recovered without external drainage.

Guisez,[11] Seiffert[12] and Kramer[13] have reported cases in which drainage of a peri-esophageal infection has been successfully carried out through the esophagoscope. In these instances the perforation was located and its edges bitten away so that free evacuation was made possible. These authors urge this procedure in "all cases of threatened mediastinal suppuration before external operation is considered."

Tucker,[14] in 1925, reported an interesting case in which a peri-esophagitis developed following the removal of a foreign body and in which he successfully drained a large abscess just below the cricopharyngeus by incision through the posterior esophageal wall.

These reports seem to indicate beyond question that patients with the most outspoken evidence of perforation and peri-esophageal infection may recover without external drainage.

REPORT OF CASES.

Case 1.—F. B., a man of 38, was admitted to the Strong Memorial Hospital because of pain in the neck and inability to swallow. Four days before, he had swallowed a fish bone. The day after the accident an unsuccessful attempt to dislodge the bone had been made by the family physician. His symptoms increased rapidly after this manipulation. The presenting symptoms and signs at the time of admission were: pain, exquisite tenderness and slight but definite swelling along the right side of the esophagus just below the level of the cricoid cartilage; complete inability to swallow. No emphysema clinically or by X-ray. Temperature 101°; leucocytosis 12,000. An esophageal speculum was passed under local anesthesia. The walls of the upper esophagus were markedly swollen. A fish bone, 3 cm. in length, was found with its point deeply embedded in the right lateral wall. After removal a short, deep laceration was clearly visible but no pus was noted. The fever subsided within the next forty-eight hours and the patient made an uneventful recovery.

Case 2.—L. M., a man of 28, admitted to the hospital because of pain in the neck and inability to swallow. Three days before, while eating a beef stew, he had noticed a sharp, stinging pain in the region of the upper esophagus. An X-ray, taken soon after the accident, was negative and his family physician advised watchful waiting. His symptoms increased steadily until he was unable to swallow anything. Examination at the time of admission showed exquisite tenderness and a slight swelling along the anterior border of the right sterno-cleido-mastoid muscle, just below the level of the cricoid cartilage. No emphysema. Temperature 101.8° F.; W. B. C. 14,500. Lateral X-rays of the cervical region showed no foreign body, emphysema or broadening of the prevertebral space. No opaque mixture was given. Under local anesthesia an esophageal speculum was gently introduced. There was marked swelling of the tissues at the introitus of the esophagus. As the speculum was advanced, a sudden gush of pus was observed. No foreign body, however, was noted. Further manipulation seemed inadvisable and it was decided to await further developments. The following day there was very evident improve-

ment in the clinical picture and this continued to complete recovery after five days.

Comment.—These two cases well illustrate the power of the esophagus to localize infection. In the first case, a localized peri-esophagitis occurred, promptly subsiding with the removal of the foreign body, and in the second case a peri-esophageal abscess which was successfully evacuated through the esophagoscope. Many similar cases have been reported in the literature. During the past year the writers have conducted a series of experiments in dogs which beautifully illustrate the protective power of the animal esophagus. In six dogs, under amytal anesthesia, a clean incision varying from 1 to 2.5 cm. in length, was made through an esophageal speculum in the lateral or porterior wall of the cervical esophagus. Aside from a short period of dysphagia, no complications developed and at postmortem study, from two to three weeks later, it was exceedingly difficult to identify the site of laceration. In another group of animals, the edges of the incision were bitten away so that a gaping perforation was effected. These dogs also recovered without evidence of neck suppuration. In a third group a small pocket was made in the peri-esophageal tissues and a pledget of cotton saturated with pus freshly obtained from a lung abscess cavity was placed snugly in it. These animals also recovered uneventfully. In this series of fourteen dogs we were unable to produce the train of events seen in perforations of the human esophagus.

Case 3.—M. P., a woman of 50, was admitted to the hospital because of sticking pain in the left side of the neck and marked dysphagia. Her symptoms had come on twenty-four hours before while eating spaghetti. There was neither swelling nor emphysema, but very marked tenderness on lateral pressure over the esophagus from the left side. The temperature was normal. The X-ray showed a common pin in the esophagus at the level of the sixth vertebra. No emphysema could be detected about its point. Fluoroscopic guidance was necessary to locate the pin, which had perforated the left lateral wall of the esophagus, with its head deeply buried in the tissues. The patient was observed for forty-eight hours following the removal, and then, because of complete absence of symptoms or signs of infection, was discharged. Four

days later she returned to the hospital complaining of pain and swelling in the left side of the neck as well as marked dysphagia. Examination showed exquisite tenderness along the anterior border of the left sterno-cleido-mastoid muscle in its middle third, moderate swelling but no palpable emphysema. A fever of 101° was present. The X-ray gave no additional evidence. The W. B. C. was 12,000. Thirty-six hours after admission, because of rising fever and leucocytosis as well as increasing severity of local symptoms, external drainage was performed. An abscess was discovered and drained beneath the left lateral lobe of the thyroid. Convalescence was entirely uneventful.

Comment.—The potential dangers of minute perforation of the esophagus deserve particular emphasis. An abscess may form slowly, and many days after the removal of the foreign body suddenly burst into the mediastinum. Wiethe[15] reports three such cases, in which the site of the original perforation could not be found at autopsy. Invasion of the carotid sheath with all its serious sequelæ is not uncommon in these cases. Myerson's[4] case, already referred to, is an example of this complication. It is imperative that all cases with minute perforations be followed for several days after successful endoscopic removal of the foreign body before being discharged from observation.

Case 4.—G. R., a woman of 45, was admitted to the hospital six hours after swallowing a fragment of chicken bone. She was suffering with considerable pain along the left side of the neck, especially at the level of the cricoid cartilage. She admitted that she had tried to dislodge the foreign body by external manipulations. There was no fever, and the chief sign on examination was exquisite tenderness over the area of pain. X-ray was negative. Under local anesthesia a sharp triangular fragment of bone was removed from the upper esophagus. At this time a deep laceration along the posterior portion of the left wall was observed. The following day there was slight, though definite, subcutaneous emphysema in the left side of the neck without external evidence of inflammation. The temperature was 100.6°. Her general condition was excellent and the pain was definitely decreased. A course of watchful waiting seemed advisable. The note made on the morning of the second postoperative day reads:

"Not so much emphysema. Tenderness not increased. The temperature is down and the patient is symptomatically improved." Late that afternoon, however, the temperature rose to 102.8°, only to fall to 100.6° at the evening reading. The W. B. C. at this time was 21,700. On the third postoperative day the highest temperature was 100°. The observer's note read: "Symptoms and signs are very definitely improved. Swallowing is decidedly easier. There is no tenderness along either carotid sheath. Emphysema has disappeared. I feel that the patient is progressing satisfactorily and that there is no apparent basis for external drainage." This opinion was also expressed by the surgical consultant. That night, however, she became suddenly worse; the pulse weak and thready. An X-ray of the chest showed definite mediastinitis. External drainage was obviously impossible. Death occurred the following afternoon.

Autopsy Findings.—The lateral spaces of the neck between the esophagus and the carotid sheaths were intact and uninvolved. Posterior to the esophagus was a track of infection leading from a cervical perforation downward to the mediastinum. A generalized mediastinitis was present with perforation through the diaphragm and formation of a subdiaphragmatic abscess. A serofibrinous peritonitis was also present.

Comment.—This case is particularly instructive because it illustrates the danger of relying on clinical evidence of improvement. It emphasizes the necessity for prompt external drainage in cases where perforation in or near the posterior wall of the cervical esophagus is known to exist. The intimate relation between the postesophageal space and the mediastinum has already been referred to. Because of the depth of the infection the external signs in these cases may be surprisingly few. Repeated lateral X-rays would have been of unquestioned value in following this case and might have indicated external drainage before it was too late.

Case 5.—A. R., a man of 69, was admitted to the hospital because of extreme pain in the left side of the neck and inability to swallow even liquids. Twenty-four hours before he had swallowed a prune pit. The temperature was 100.6°. There was exquisite tenderness on pressure over the left wall of the esophagus

just below the cricoid level. There was no demonstrable emphysema. Under local anesthesia, a prune pit was a needle-like point was removed from the upper esophagus. A deep laceration, 1½ cm. in length, was noted in the left lateral wall at the site of lodgment. Four hours later there was palpable evidence of emphysema in the left side of the neck. The following morning emphysema had increased, there was exquisite tenderness along the anterior border of the left sterno-cleido-mastoid muscle, and the temperature had risen to 102.8°. The W. B. C. was 18,000. External drainage was considered urgent. The procedure carried out consisted of exposure of the left lateral wall of the esophagus by means of an incision along the anterior border of the sterno-cleido-mastoid muscle and packing of this area "in order to wall off the mediastinum." In spite of vigorous supportive treatment, the patient died four days later.

Autopsy revealed a perforation of the cervical esophagus with infection spreading along the postesophageal space to the mediastinum. Phlegmonous mediastinitis, fibrous pericarditis, empyema and bronchopneumonia were present.

Comment.—This case illustrates the importance of walling off not only the lateral but also the postesophageal fascial space in the course of external drainage operations. It is reasonable to assume that proper appreciation of this fact might have saved this patient from fatal extension to the mediastinum.

Case 6.—G. K., a man of 56, was seen five hours after an esophagoscopy had been done for diagnostic purposes. It was stated that the examination had been a difficult one and that considerable bleeding had caused the operator to discontinue the examination. The patient appeared in shock with rapid, weak pulse and low blood pressure. There was marked pain in the left neck radiating downward to the shoulder, exquisite local tenderness and subcutaneous emphysema. The temperature was 102.8°. Immediate external drainage was advised and done in accordance with the technic subsequently described. No attempt was made to identify the perforation, but the visceral and postvisceral fascial spaces were carefully drained. Recovery was uneventful. Two weeks later, by exploratory laparotomy, an inoperable carcinoma of the cardiac end of the stomach was found.

Comment.—This case illustrates the possibility for success in the management of instrumental perforation of the cervical esophagus by prompt external drainage.

Case 7.—E. F., a woman of 42, was admitted to the hospital with the history of having swallowed a chicken bone two hours before. An unsuccessful effort had been made by the family physician to dislodge it by means of a bougie. There was no fever or external changes except tenderness or lateral pressure over both sides of the cervical esophagus. An X-ray showed the foreign body at the level of the sixth cervical vertebra. Because of the extreme nervousness of the patient, endoscopy under ether anesthesia was agreed to. During the induction she vomited up a large, flat, triangular fragment of chicken bone. The cervical esophagus was inspected through a speculum and a deep laceration was noted on each lateral wall at the apparent site of the lodgment. The patient left the hospital forty-eight hours later in apparently excellent condition. Two days later she returned because of recurrence of neck pain. The admission temperature was 101.8°. There was exquisite tenderness along the anterior border of the right sterno-cleido-mastoid muscle but no swelling or emphysema was noted. W. B. C. 18,000. The lateral X-ray was inconclusive. Rather than risk the possible dangers of expectant treatment, external drainage was decided upon. "The peri-esophageal structures below the level of the inferior thyroid artery were indurated but no free pus was encountered. There was emphysema of these tissues, and after their separation air was extruded with each respiration." Drainage of the lateral and postesophageal fascial spaces was carried out. Convalescence was uneventful and the patient was discharged from the hospital on the twelfth postoperative day.

Comment.—This case falls in the group which, in the opinion of many observers, might well be managed expectantly. We frankly feared the responsibility of watchful waiting and elected a course which seemed to assure safety by controlling possible extension along the fascial planes. The result, we feel, justified the course elected.

Case 8.—E. H., a young woman of 22, experienced severe pain in the throat while eating clam chowder. The pain soon centered

Fig. 1. Oblique view of cervico-thoracic region in case No. 8. The deep emphysema is shown. There was no palpable subcutaneous emphysema.

just above the episternal notch and became increasingly severe so that when she entered the hospital, twenty-six hours later, even liquids could not be swallowed. Examination revealed exquisite tenderness anterior to the lower portion of the sterno-cleido-mastoid muscle on the right, but no evidence of emphysema or swelling. The temperature was 101.6°. W. B. C. 22,000. When a lateral X-ray (Fig. 1) was taken air could be seen

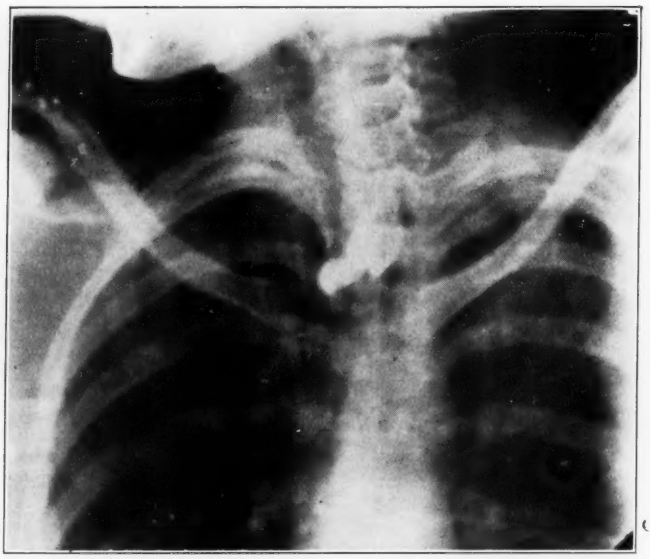

Fig. 2. The opaque media is extravasated into the neck. At operation it was found to have spread out through the tissues for a greater area.

in the peri-esophageal tissues on the right side, and after a swallow of lipiodol was given this space could be seen filled with the extravasated fluid. (Fig. 2.) An esophageal speculum was cautiously introduced and a deep laceration on the right lateral wall was identified. No evidence of foreign body, however, was seen. Preparations were then made for external drainage, which was done soon afterwards. The patient, at best a thin, nervous girl, was in very poor condition, with a pulse rate of 140. Under regional block and local infiltration, the usual exposure of the esophagus was effected from the right side. A perforation one and one-half inches long was clearly visible in the lateral wall of the esophagus, beginning just at the level of the cricoid cartilage. (Fig. 3.) The opening was closed with interrupted catgut sutures. The lateral and postesophageal spaces were carefully packed off with gauze drains. The patient

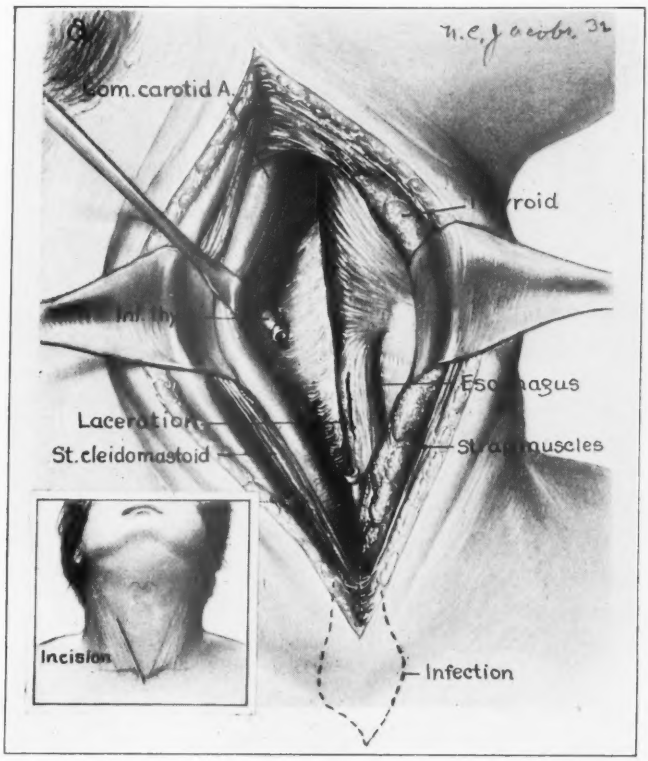

Fig. 3. The esophageal laceration in Case No. 8. For the sake of clarity the incision is enlarged in the illustration. The actual length of the incision is shown in the insert. The exposure obtained by the medial mobilization of the thyroid gland is well shown. The dotted area represents the limits of gravitation of the infected material.

stood the operation well. On the following day a gastrostomy was done under local anesthesia. On the first postoperative day the girl was quite ill with a fever of 103.6°, pulse 135, respiration 40, but from that time on she gradually improved, and on the eighth postoperative day all drains were removed except for a small superficial one. On the twelfth day a chest X-ray (Fig. 4) showed a high right diaphragm, slight cloudiness at the right base, sug-

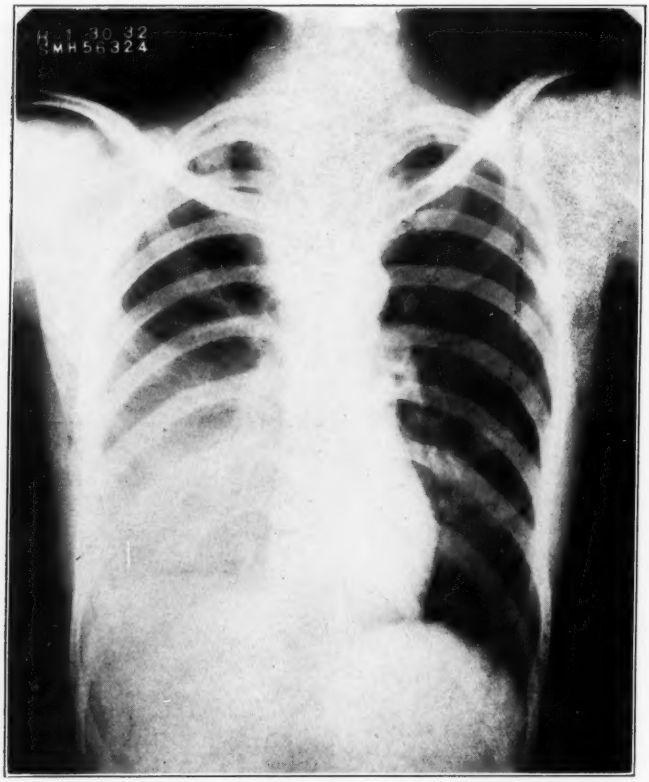

Fig. 4. Chest X-ray in Case No. 8 taken on twelfth postoperative day showing the high right diaphragm, cloudiness at the right base suggesting fluid and an area of increased density in the superior mediastinum.

gesting fluid, and an area of increased density in the superior mediastinum, suggesting abscess formation. Three days later (the fifteenth postoperative day) a catheter was inserted through the lower angle of the wound and worked downward into the superior mediastinum. Thirty cc. of pus were exacuated by suction. Dakin's irrigations were carried out daily, and an X-ray taken eight days later showed a disappearance of the mediastinal

shadow. Five days later the catheter was removed. On the twenty-seventh postoperative day fluids by mouth were resumed cautiously. One week later, thirty-four days after operation, the gastrostomy tube was removed. The patient was discharged on the fortieth postoperative day, at which time the wounds were healed and she was taking a solid diet without difficulty. An X-ray of the chest at this time (Fig. 5) reported a very marked clearing of all previous shadows." This girl has been carefully followed for four months following the discharge from the hospital and has developed no subsequent trouble.

Comment.—This case illustrates the successful management of an unusually large perforation of the cervical esophagus, which most certainly would have proved fatal had any delay in instituting radical measures been attempted. Actual involvement of the superior mediastinum occurred and was successfully drained through the cervical approach. The case is the more interesting because of the poor general condition of the patient when surgical intervention was decided upon. This case also emphasizes the importance of the X-ray in determining the progress of an infection in the cervical region.

TECHNIC FOR EXTERNAL DRAINAGE.

The external operation for cervical esophageal perforation was originally described by Marschik.[7] The procedure, which has not been essentially modified to date, has twofold purpose, namely, blocking of the fascial spaces to the mediastinum and draining of the extravasated material. This is accomplished by packing gauze between the vascular sheath and the trachea and esophagus low in the neck to obliterate the anterior fascial space.

Then by retracting the thyroid medially the space behind it and beside the esophagus is packed. This is to block the posterior path to the mediastinum. During this latter maneuver, pus or extravasated material may be encountered and drained. The esophagus is undisturbed.

Palmer[16] describes the Marschik operation as he learned it in Hajek's clinic in Vienna as follows: An incision is made from the mastoid to the sternum. The sterno-cleido-mastoid muscle is retracted "until the vascular sheath is reached." This is called

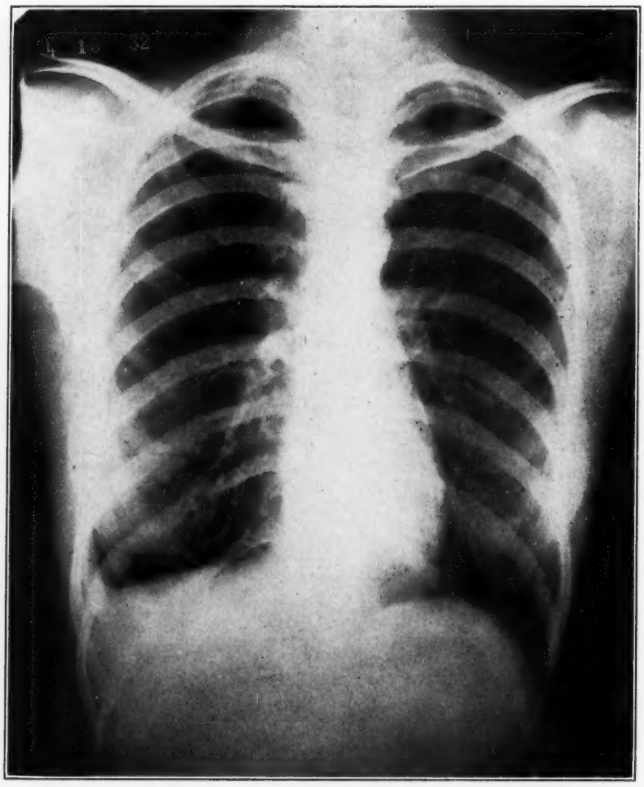

Fig. 5. Chest X-ray in Case No. 8 taken three months later showing complete clearing of chest pathology.

the anterior collar mediastinum and is tamponed with iodoform gauze. The dissection is carried upward, the omohyoid is cut and "by gently lifting the thyroid forward with blunt retractor the posterior collar mediastinum is reached. The index finger may now be inserted until the cervical vertebræ are encountered." This area is tamponed and the original source of the infection is sought if the operator desires. The same description is given by Keiper.[17] No mention of the operation was given in the modern textbooks

or systems of surgery or otorhinolaryngology that were consulted.

The operation as originally performed in this clinic differed in a few details but not in essential principles. A smaller incision was used, either transverse or parallel to the anterior border of the sterno-cleido-mastoid muscle just above its clavicular attachment. The fascial spaces between the carotid sheath and the trachea, thyroid and esophagus were packed down to the vertebral column. The omohyoid was not cut. The carotid sheath was occasionally opened and drained. This was thought to give obliteration of the paths to the mediastinum with minimum external scar. The postmortem dissection in cases Nos. 4 and 5 demonstrated conclusively that infection could track down behind the esophagus without involving the areas mentioned. In case No. 5, the prophylactic operation, as previously described, was done without benefit. From this experience it was obvious that the Marschik operation or its modifications was incomplete in so far as it did not block the space behind the esophagus.

The technic of the operation as now performed is as follows: A three-inch operation is made parallel to the clavicular attachment of the anterior border of the sterno-cleido-mastoid muscle. The carotid sheath is identified and retracted laterally with the muscle. The thyroid gland is exposed and retracted medially. The middle thyroid vein is ligated and divided. This gives more mobility to the thyroid and allows exposure of the inferior thyroid artery as it comes beneath the carotid sheath. This artery is then ligated and divided if necessary, after which the thyroid gland may be rotated medially to the midline, exposing the trachea and esophagus. The cleavage plane between the prevertebral fascia and the esophagus is outlined and the finger inserted behind and beyond the esophagus lifting it free of this space. Gauze or a drain is inserted with the tip extending to the opposite side the esophagus and brought out behind this structure, then lateral to the esophagus, trachea and thyroid gland and medial to the carotid sheath and sternomastoid muscle. (Fig. 6.) If the symptoms point to unilateral involvement the operation is terminated. If bilateral extravasation is suspected a similar incision is made on the opposite side and a drain inserted down to meet that already placed. Thus

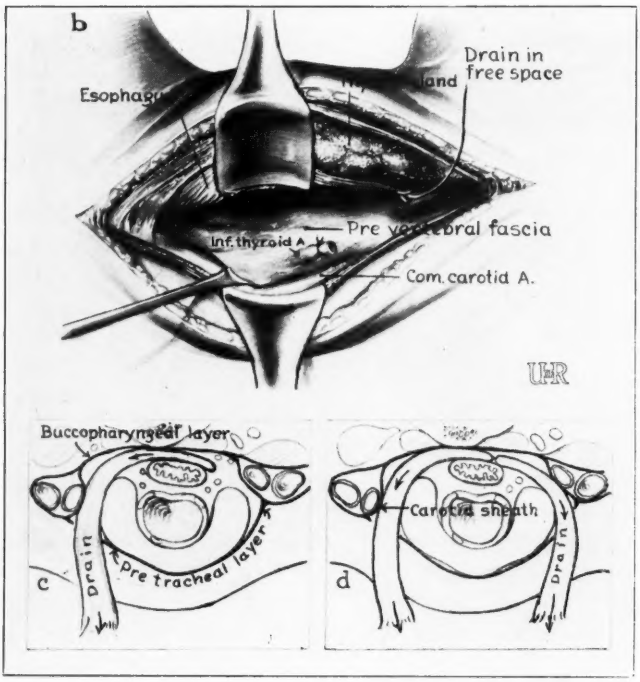

Fig. 6. Shows the free space developed by lifting the esophagus off the prevertebral structures. It is through this space that material from a posterior perforation gravitates to the mediastinum. The cross sections *c* and *d* show how the facial spaces are blocked to prevent dependent drainage into the mediastinum.

the visceral space is encircled and all paths of spread to the mediastinum effectively blocked.

This operation is done under local anesthesia after regional block of the cervical nerves at the posterior border of the sternocleido-mastoid muscle. A small amount of intracutaneous infiltration may be used in the line of the skin incision. The operation does not cause shock. Case No. 8, who was moribund when brought to the operating room, showed no perceptible change in pulse, blood pressure or general condition following the procedure. If done under local anesthesia, by a competent surgeon,

it should not involve more risk than the average operation for goiter, which Lahey[18] has shown to have a mortality of 0.5 per cent.

CONCLUSIONS.

First, there is much evidence to prove that perforation of the esophagus may occur without the formation of pus. An esophagitis or peri-esophagitis occurs, followed by a local reaction of varying severity, and may subside either with the removal of the foreign body or shortly thereafter. Case 1 illustrates this type of perforation.

Second, there are many cases on record which prove that nature may so effectively wall off infection following perforation that evacuation into the esophagus may occur either at the time of removal of the foreign body, during esophagoscopy, even when no foreign body is discovered, or shortly afterward. Case 2 illustrates this variety. Furthermore, such well localized infection may present in a position favorable for instrumental drainage through the esophagoscope, as is well illustrated by the cases of Guisez,[11] Seiffert,[12] Kramer[13] and Tucker.[14]

Third, emphysema, although positive evidence of perforation, cannot be considered as indication that recovery cannot occur without external drainage. Jackson,[19] Myerson,[4] von Eicken[9] and other observers have described cases of extensive emphysema in association with marked local and general disturbances that have recovered under conservative treatment. This fact, however, does not, in our opinion, indicate that surgical noninterference is the procedure of choice. Case No. 4, in which extensive emphysema was disregarded and after a period of very definite clearing of all external signs, sudden overwhelming mediastinitis occurred due to sinking of the infection in the postesophageal spaces, could probably have been saved by prompt external drainage. This case illustrates clearly the impossibility of judging accurately from the clinical signs the progress of a peri-esophageal infection as well as the relative danger of mediastinitis. Case No. 8, presenting emphysema as the result of an extensive tear of the cervical esophagus, could never have been saved except by immediate external drainage.

The fact that certain patients with high fever, extensive emphysema, marked swelling and tenderness recover without surgical intervention is, in our opinion, no more argument against the advisability of early external drainage than is the well known frequency of recovery from outspoken signs of acute appendicitis a valid objection to early appendectomy. Surgeons have come to agree that it is far better to operate too frequently in these cases than to run the risk of watchful waiting. Deaver's[20] summary of this attitude seems to us peculiarly applicable to the subject under discussion. "Candor and fact compel the admission that many, perhaps even a majority of cases of appendicitis, will recover as a result of the defensive powers of nature unaided by treatment of any kind. To admit this is but to testify to the great protective and healing power of nature. On the other hand, nothing is better substantiated than that a very considerable portion go on to complication. It is utterly beyond the skill or wisdom of any man to predict a favorable outcome in any case by means other than operative." We feel that the analogy between perforations of the cervical esophagus and acute inflammation of the appendix is a close one. Better to operate early, even though recovery might occur under conservative management, than to risk unduly a fatal extension to the mediastinum.

We acknowledge the necessity of considering each case as an individual problem, as well as the difficulties of formulating exact rules of management. Under appropriate circumstances, conservative measures or endoscopic drainage may be attempted, but if prompt improvement does not occur we urge early external drainage. We would submit the following indications for external operation:

1. Cases presenting definite evidence of localized abscess in the neck.

2. In all cases where gross perforation is known to have occurred as the result of faulty instrumentation, prompt external drainage is imperative. The few cases recovering without surgery do not weaken this contention.

3. In cases presenting X-ray evidence of a foreign body outside the lumen in the peri-esophageal tissues, as in the case reported by Iglauer and Ransohoff.

4. In cases where perforation of the posterior esophageal wall can be established by endoscopic or X-ray study, we consider it safest to institute early external drainage because of the intimate association of the postesophageal space with the mediastinum as well as known unreliability of the symptoms and signs in such cases.

5. Emphysema, demonstrable clinically or by X-ray, in association with rising fever and leucocytosis, indicates a serious situation which makes external drainage advisable.

6. Finally, in any case when in the presence of a known or suspected perforation any reasonable doubt arises as to the safety of the patient, we urge prompt external drainage as a procedure without undue risk and best suited to control a situation which at any moment may otherwise become uncontrollable.

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BIBLIOGRAPHY.

1. Saint, J. H.: Surgery of the Esophagus. Arch. Surg., 19:53, 1929.

2. Furstenberg, A. C.: Tr. 35th meeting Am. Laryng., Rhin. and Oto. Soc., p. 210, 1929.

3. Killian, H.: Beitrag zur operativen Behandlung komplizierte Fälle von Fremdkerpern der Speiserohre mit besonderer Berücksichtigung der Indikationen. Arch. f. Klin. Chir., 122:382, 1923.

4. Myerson, M. C.: Perforation of the Esophagus. Annals of Otology, Rhinology and Laryngology, 37:545, 1928.

5. Iglauer, S., and Ransohoff, J. L.: Perforation of the Esophagus by a Foreign Body, with Report of a Case Presenting Unusual X-ray Signs. Laryngoscope, 34:821, 1924.

6. Schlemmer, F.: Erfahrungen mit Ösophagus-Freundkörpern in einem Zeitraume von zehn Jahren. Arch. f. Klin. Chir., 114:37, 269, 1920.

7. Marschik, H., and Vogel, H.: Fremdkörper in dem oberen Luft- und Speisewegen mit besonderer Berücksichitigung der Ösophagotomie. Wiener klin. Wochns., 22:1405, 1909.

8. King, E.: Perforation of the Esophagus with Report of Six Cases. Annals of Otology, Rhinology and Laryngology, 38:351, 1929.

9. von Eiken: Zur Frage der Verletzung des Hypopharynx und des obersten Ösophagus-abschnittes durch Fremdkörper. Zeitsch. f. Hals-Nasen-u-Ohren., 15:387, 1926.

10. Hajek: Aussprache zu Vortrag. Zeit. f. Hals-Nasen-u-Ohren., 15: 392, 1926.

11. Guisez, J.: Considerations sur nas differents cas de corps etrangus de l'oesophage et des bronches dans ces dix dernieres annees. Bull. Oto.-Rhino.-Laryn., 23:221, 1925.

12. Seiffert: Zur Erkennung und Behandlung von Ösophagus-perforationen. Zeit. f. Hals-Nasen-u-Ohren., 12:290, 1925.

13. Kramer, R.: Endoscopic Treatment of Esophageal Suppuration. Laryngoscope, 39:97, 1929.

14. Tucker, G.: Retropharyngeal Abscess from Lodgment of Foreign Body in Esophagus. J. A. M. A., 84:511, 1925.

15. Wiethe, C.: Über Ösophagus-perforationen herausgerufen durch Ösophagoskopie. Zeit. f. Hals-Nasen-u-Ohren., 28:58, 1930.

16. Palmer, F. E.: Descending Abscess in the Neck. J. A. M. A., 83: 2067, 1924.

17. Keiper, G. F.: Abstract of Discussion. J. A. M. A., 85:99, 1925.

18. Lahey, F. H.: The Crisis of Exophthalmic Goiter. New Eng. Jour. Med., 199:255, 1928.

19. Jackson, C.: Foreign Bodies in the Air and Food Passages. Tr. 29th meeting Am. Laryn., Rhin. and Otol. Soc., p. 441, 1923.

20. Deaver, J. B., and Pfeiffer, D. B.: Appendicitis. Keen's Surgery, Saunders, 1922, Vol. 8, p. 439.

CI.

THE ROENTGEN TREATMENT OF PRIMARY MALIGNANT DISEASE OF THE TRACHEOBRONCHIAL TREE.

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ROCHESTER, MINN.

Many theories have been advanced in the last decade as to the reasons for the striking increase in the incidence of primary malignant disease of the tracheobronchial tree. There is no question that the more frequent antemortem recognition of such lesions has been due almost entirely to the more general use of the bronchoscope in the diagnosis of pulmonary affections. The bronchoscopist has learned through experience the gross similarity of many different types of lesions in the tracheobronchial tree and the necessity for removing tissue for microscopic study from every area that appears suspicious. The perfection of this bronchoscopic procedure has made it possible to recognize malignant disease in the trachea and bronchi at a reasonably early stage, and advancement in diagnosis has brought with it the responsibility for suggesting some method of palliation or cure.

Direct attack on the lesion by fulguration or diathermy through the bronchoscope seems ill advised in the majority of cases because of the fact that the disease usually involves one of the main bronchi and extends well beyond the confines of the bronchial wall. As a matter of fact, the lesion is actually much more extensive than would appear on bronchoscopic examination, although it is usually smaller than one would conclude from roentgenoscopic study. Surgical removal of bronchogenic carcinoma has been accomplished on several occasions by a limited number of operators, but because of the situation of the lesion one would scarcely expect to obtain satisfactory results from operation frequently. Another method of treatment applicable in the disease

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is the employment of radium or deep roentgen rays. Although radium emanations in the form of radon seeds may be introduced without difficulty into bronchial carcinoma, we have preferred to use the roentgen ray in treatment.

The technic of applying the roentgen rays now in use in the Section on Therapeutic Radiology of The Mayo Clinic is the result of a slow process of evolution, and has developed more from a clinical than from a biologic or physical point of view. At the onset, treatment was given with the utmost conservatism, owing to the fear which subsequently proved to be ungrounded, of producing roentgen injury to the lungs and structures in the mediastinum, and owing to failure properly to select cases for treatment. Encouraged by the demonstration that the lungs will tolerate doses of roentgen rays which are within the limit of tolerance for the skin, and because of more thorough knowledge of pleuropulmonitis from the basic work of Desjardins, treatment is now given without hesitation to many patients to whom it was denied in the early days.

Since all tumors of the parenchyma of the lung and the bronchi are deep seated and of relatively high radioresistance, it is self-evident that treatment must be carried out at high voltage and that the principle of cross fire must be adopted. Even though the absorptive characteristics of pulmonary tissue are at present imperfectly understood, it has been proved that a four field cross fire with one field through each hemithorax anteriorly and posteriorly with the vertical beams directed into the lesion not only is well tolerated by the average patient but produces favorable clinical results. Whether an increase of dose by applications of radium in situ may lead to better results is at present an unsettled problem, as theoretically the danger of producing radium necrosis of the bronchial wall is one that must be given serious consideration.

With the facilities at our disposal the treatment of each field is carried out as follows: The roentgen rays are generated at 200 kilovolts and 5 milliamperes by a transformer with mechanical rectifier activating an air-cooled high voltage Coolidge tube. The irradiation emerging from a Failla drum tube-holding device is filtered through 0.75 mm. copper and 2 mm. aluminum. With

this set-up and at a target skin distance of 50 cm., the threshold erythema dose is produced in seventy minutes, and, measured in air by the Victoreen dosage meter, equals 560 r. We have had no experience with constant potential machines nor have we tried any of the methods of protracted or fractionated irradiation.

The tumor having been localized both by bronchoscopic and roentgenographic examination, the administration of the necessary cross fire of the region is facilitated. Extreme care is used in arranging the patient and roentgen ray tube to ensure a correct direction of the vertical beam into the lesion. To avoid any chance of missing the tumor large fields are used with each vertical beam at an angle of about 45°.

Since some patients are extremely uncomfortable from dyspnea and irritating cough, it is essential to minimize systemic reactions and avoid undue fatigue by dividing the treatment (total dose) into sessions of moderate duration. Most patients can tolerate complete irradiation of one of the four fields each day. In a few cases it is necessary to divide the dose for each field into two or more sessions. A complete course of treatment, therefore, may require from four days to two weeks, depending on the patient's condition and ability to tolerate irradiation.

The average patient takes one field for each of four days without undue reaction. Nausea and vomiting are seldom severe enough to require care, and are reduced to a minimum by the patient's abstinence from food for three hours before and after treatment. After four fields of treatment a drop of blood pressure or destruction of the blood elements has not been sufficient to cause any concern.

Shortly after the subsidence of the immediate roentgen reaction the patients notice increased expectoration, sometimes of frankly purulent material, and a subsidence of fever, followed in a few days by cessation of cough and decrease of pain and dyspnea. In all probability these effects are due to an action of the rays on the inflammatory infiltrate and mucus producing glands which surround the lesion. The radiosensitivity of these structures has been established by abundant clinical and laboratory experiments. In about two weeks regression of the tumor begins and the symptoms abate as regression proceeds. When these

changes are slight the dose effective in the tumor was too low. Improvement reaches its maximum about a month after the roentgen ray treatment and has almost run its course by the end of the sixth week. We feel, therefore, that symptomatic improvement, no matter how slight, indicates repetition of treatment, as it is almost invariably accompanied by organic improvement in the malignant process. The technic of the second course of treatment given about six weeks after the first, although essentially similar to the first course, is modified to suit the data obtained by bronchoscopic and roentgenographic examination. If conditions indicate, the second course is followed by a third course of roentgen therapy after an interval of two months.

From May 1, 1925, to January 1, 1931, seventy-one cases of primary malignant disease of the tracheobronchial tree were diagnosed at The Mayo Clinic by the removal of tissue from the wall of the bronchus through a bronchoscope. We have been able to keep in close touch with all of these patients, and this report deals especially with those receiving beneficial results from deep roentgen-ray treatment.

Sixty-one of the patients are dead, and of this number thirty-two received treatment at the clinic or at home. Those receiving treatment lived from two weeks to four years after returning home, the average duration of life being slightly more than eight months.

Twenty-nine, or all untreated patients, lived from a few days to thirty-one months after a positive diagnosis had been made, or an average of a little more than five months. This difference in the duration of life with and without treatment may have been due partly to the fact that certain of the latter group were not treated because of complications that made irradiation inadvisable. In most cases, treatment was offered to patients regardless of the existence of pleural effusion or metastasis.

It is difficult, if not impossible, to determine the duration of symptoms of pulmonary carcinoma, as it is likely that pulmonary abscess or bronchiectasis has antedated many cases of malignant disease of the lungs. However, the duration of pulmonary symptoms in the treated and untreated cases was approximately the same. One patient had radon seeds implanted into the bronchial

growth after he left the clinic; he survived for four years. Two patients observed since January, 1931, have been treated by implantation of radon seeds in the bronchial lesion, but neither showed evidence of beneficial effect. Ten patients who were treated by deep roentgen rays are improved or are living and well from fifteen months to four years after treatment. Eight of this group received treatment under our direction and two were treated after returning home.

ABSTRACTS OF CASES.

Case 1.—A woman, aged forty-one years, was examined February 3, 1928. She had had an attack of pneumonia in infancy and had always had more or less discomfort in the right thoracic region. In 1918 an attack of influenza was followed by copious pulmonary hemorrhages, and since then she had had intermittent cough, and expectoration of large amounts of purulent sputum. During the two years previous to her examination at The Mayo Clinic, she had noted increasing dyspnea and much pain in the right side of the thorax and frequent hemoptysis.

Examination of the thorax revealed a dull percussion note and distant breath sounds at the base of the right lung. Roentgenograms demonstrated localized atelectasis at the base of the right lung with a small pleural effusion, and the presence of bronchial carcinoma was questioned. Bronchoscopic examination revealed an infiltrating ulcerating lesion in the bronchus to the lower lobe of the right lung, and on removal of tissue for microscopic examination adenocarcinoma, graded 4, was reported. The patient was given a course of treatment and was permitted to return home. August 21, 1931, she returned for reconsideration, and although she continued to have a slight cough and small amounts of purulent sputum, she had gained thirteen pounds in weight and considered her condition satisfactory. There had been very little change in the pulmonary lesion. Her general condition was still good, February 22, 1932, the date of her last report.

Case 2.—A man, aged fifty-six years, was examined August 9, 1929. He had been well until December, 1928, when a respiratory infection developed that lasted for three weeks. A similar attack was experienced in February, 1929, and a third attack in March, followed by sore throat and cough, lassitude, loss of thirty pounds in weight, dyspnea and wheezing in the thorax The wheezing was particularly troublesome when he was lying on the right side.

From general and roentgenoscopic examination it was thought that the pulmonary lesion was bronchiectasis at the base of the right lung. On bronchoscopic examination the mesial wall of the right main bronchus was considerably infiltrated and on removal of tissue for microscopic examination adenocarcinoma, graded 2, was reported. Roentgen ray treatment was followed by complete relief from symptoms. A letter from the patient's physician, dated January 29, 1932, stated that he was well except for slight dyspnea on exertion.

Case 3.—A woman, aged twenty-five years, came for examination September 9, 1929, complaining of attacks of severe pain situated in the right lower abdominal quadrant radiating to the right costal margin. The pain had been present for about a year, coming on at intervals of three weeks and usually appearing a week before menstruation. The attacks were associated with elevation in temperature, required hypodermics for relief and were frequently accompanied by nausea and vomiting. During the few weeks preceding her visit at the clinic she had noticed a little cough and dyspnea on exertion. There has been a loss of twenty pounds in weight. Abdominal exploration had been advised and she came to the clinic for operation.

Examination of the thorax revealed infiltration at the base of the right lung, and this was confirmed by roentgenographic observation. Bronchoscopic examination revealed an ulcerating infiltrating lesion in the right main bronchus. Tissue removed for microscopic examination proved to be adenocarcinoma, graded 3. Roentgen ray treatment was given. February 10, 1932, the patient reported that she had been perfectly well except for a slight nonproductive cough.

Case 4.—A man, aged sixty-four years, was examined April 29, 1930. He had been well until November, 1929, when he contracted a cold, severe enough to require hospitalization for a period of ten days. This was followed by a cough productive of sputum, but he felt well enough to return to work until January 15, 1930, when a chill with fever developed. From that time, it was thought he had recurring attacks of pneumonia. He had lost thirty-five pounds in weight.

General and roentgenoscopic examination of the thorax revealed infiltration in the lower lobe of the right lung with definite evidence of bronchostenosis. Bronchoscopic examination revealed a large fungating tumor in the bronchus to the lower lobe of the right lung. Tissue was removed for microscopic study and squamous-cell epithelioma, graded 4, was found. Roentgen ray treatment was given and at the time of the patient's last report, in November, 1931, his general condition was satisfactory, although he continued to have some cough with the expectoration of mucoid secretion.

Case 5.—A woman, aged thirty years, was examined April 14, 1930. In 1927 following a miscarriage a peculiar substernal discomfort had developed, and a month later she coughed up a tiny streak of blood. A year later she experienced two attacks of strangulation that were thought to be asthmatic. A change of climate was followed by complete relief from symptoms until January, 1929, when during the course of an acute respiratory infection she expectorated a moderate amount of blood and noted attacks of dyspnea. From then until her examination at the clinic, blood was expectorated on several occasions and dyspnea on exertion became pronounced.

Examination of the thorax revealed an area of infiltration at the base of the right lung, and roentgenograms showed a shadow at the region of the right hilum. The bronchus to the lower lobe of the right lung was occluded with a nonulcerating tumor, which was diagnosed microscopically as adenocarcinoma, graded 3. After a course of roentgen ray treatment, the patient returned home but came back for further consideration March 28, 1931. At this time the conditions were practically the same as at first

examination, but the patient had been completely relieved from all pulmonary symptoms. A second course of irradiation was given, and at the time of the last report, February 25, 1932, she felt perfectly well.

Case 6.—A woman, aged sixty years, was examined June 10, 1930. Her complaints were weakness, loss of weight and pains in the lower extremities.

There were no symptoms referable to the respiratory system, but roentgenoscopic examination revealed dense infiltration in the region of the right hilum. Bronchoscopy revealed a bleeding mass in the bronchus to the lower lobe of the right lung, and tissue removed from this lesion was adenocarcinomatous, graded 3. Enlarged, firm lymphatic nodes in the right supraclavicular region were thought to be metastatic. Following roentgen ray treatment at home, the patient has been reasonably comfortable, although from her last report, in March, 1932, it is probable that the size of the metastatic lymph nodes had increased and that there is metastasis to the abdomen.

Seventeen and ten years before the patient's examination at the clinic both breasts had been removed, and it is possible that the case represents the ulceration of metastatic pulmonary nodules into the bronchus, although the character of the disease in the breasts could not be determined.

Case 7.—A woman, aged thirty years, came for examination July 26, 1930, complaining of a chronic cough productive of sputum for two and a half years. The illness began with an attack of pneumonia and she was confined to bed for four weeks. After this she did not regain strength. A year later the cough became more severe, the sputum was stained with blood and there was a daily elevation of temperature. The symptoms continued without change until April, 1930, when dyspnea became marked and it was necessary to perform tracheotomy. Soon after this the breathing improved and the tracheal tube was removed. The tracheotomy wound was permitted to close but had to be reopened in two months because of increasing obstructive dyspnea.

Examination of the thorax did not reveal the character of the lesion. Roentgenograms disclosed an infiltration throughout both lungs of an indeterminate nature. On bronchoscopic examination, multiple nodules were found throughout the tracheobronchial tree which were thought to be papillomas. Several of these nodules were removed for examination and were reported to be squamous-cell epithelioma, graded 3. Following a single course of roentgen ray treatment at her home, the patient recovered completely except for a persistent tracheal fistula. The date of the last report was February 23, 1932.

Case 8.—A man, aged sixty-three years, came to the clinic September 1, 1930. For many years he had suffered from frequent infections of the upper part of the respiratory tract but in other respects he had been well until April, 1929. The onset of his illness was rather sudden with cough, and pain in the lower right thoracic region, and expectoration of small amounts of blood. Following this he continued to have cough with expectoration of large amounts of purulent sputum and occasional elevation of temperature. There had been a loss of weight at the onset of the illness but this was regained later. Lipiodol had been injected into the trachea one month before examination.

Examination of the thorax revealed infiltration at the base of the right lung with marked diminution in breath sounds in the same situation. Roentgenograms appeared to reveal areas of bronchiectasis in the lower lobes of both lungs. Lipiodol was still present on both sides. Bronchoscopic examination revealed an infiltrating, ulcerating, bleeding lesion in the right main bronchus, and microscopic examination of tissue removed from the growth disclosed adenocarcinoma, graded 3. A course of treatment was carried out under our direction, and a second series of irradiation was given at the patient's home with considerable improvement in his condition. In December, 1931, a roentgenogram of the thorax showed that there had not been any change in the appearance of the pulmonary lesion.

Case 9.—A man, aged forty-one years, came for examination November 17, 1930, complaining of cough of six months' duration. At first, the cough was nonproductive but later became associated with a small amount of mucoid sputum. There had been a loss of twenty pounds in weight and considerable loss in strength. Thoracentesis at home had been productive of a small amount of blood-tinged fluid.

A lesion was found in the upper lobe of the right lung with marked stenosis of the bronchus to this area. The bronchus to the upper lobe of the right lung was found to be completely occluded by a necrotic tumor. Microscopic examination of the tissue showed it to be a squamous-cell epithelioma, graded 3. Roentgen ray treatment was given and the patient was still showing gradual improvement at the time of his last report, in February, 1932.

Case 10.—A man, aged thirty years, was examined December 22, 1930. For several years he had had one or two colds during the winter which were frequently associated with expectoration of blood-stained purulent sputum. Four months before coming for examination he had a cold which was followed by cough, fever, malaise, loss of weight and the expectoration of large amounts of thick purulent sputum. On one occasion he had coughed up a considerable amount of blood.

Examination revealed infiltration in the lower lobe of the right lung which was thought to be due to bronchiectasis. Bronchoscopy revealed a large fungating tumor in the bronchus to the lower lobe of the right lung, and tissue removed for microscopic study was found to be adenocarcinoma, graded 3. The first course of roentgen ray treatment was completed January 5, 1931, and January 29 the patient returned for reconsideration. His general condition had improved to a marked degree. A second course of treatment was given at that time. This was repeated in April, 1931, and the fourth course of treatment was given in May, 1931. General improvement was progressive with a corresponding improvement in the roentgenographic appearance of the pulmonary lesion. In April, 1932, the patient was in good health.

SUMMARY.

From May, 1925, to January, 1931, seventy-one cases of primary malignant disease of the tracheobronchial tree were diagnosed by the removal of tissue from the lesion through a bronchoscope.

Twenty-nine patients did not receive treatment and all of them are dead, the average duration of life being a little more than five months.

Forty-two patients were treated by radium or roentgen rays either under our direction or at their homes. Thirty-two of these lived for an average of eight months after positive diagnosis had been made. Ten patients who received roentgen-ray treatment alone are living from fifteen months to four years after malignant tissue had been removed from the lesions. The average duration of life has been twenty-three months. Abstracts of these ten cases are presented.

PRESENTATION OF SPECIMENS AND INSTRUMENTS.

COSTOPHRENIC BRONCHOSCOPE.

CHEVALIER JACKSON, M. D.,

PHILADELPHIA.

In an old book written by me, I believe in 1901, there is an illustration of a tube to be passed inside of a bronchoscope or esophagoscope. The reason for devising the instrument was this: as you know, sometimes when we go down with the bronchoscope or esophagoscope we are in the position of the bulldog whose chain just won't let him reach the bone. You wish to enter a passage too small to admit the tube-mouth. The inner tube is put in to reach further. The costophrenic bronchoscope is also useful when a pin gets in the costophrenic angle. It is a long way down. It can be taken out through the mouth with the costophrenic bronchoscope. When I get to the pin and find not the point but the shaft I crumple the presenting part in the tube's mouth with the pin-bending forceps. Care is necessary not to go through into the pleura. The method is not for use with needles or hard steel pins; the point-sheathing method is best for these. The tube can be used with the laryngoscope or the bronchoscope. Parallel instruments for use in the esophagoscope are the same except they are without holes. In the esophagus the purpose is to go through the stricture. If you can dilate the stricture you can get the small inner tube through. In the esophagus we often need to see if there is a lesion down below.

Chevalier Jackson Costophrenic Bronchoscope.

DISCUSSION.

DR. IMPERATORI: Instead of using the Jackson suction tube and evacuator, I have found it more convenient simply to use the suction tube part of this device. It is very satisfactory in esophageal work and passes through the 53 cm. esophagoscope.

Suction tube.

AN INFANT ESOPHAGOSCOPE AND GASTROSCOPE.

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

Recently there have come under my care at the University of Pennsylvania Bronchoscopic Clinics, several cases of foreign body in the stomach and esophagus in infants under six months of age. Three of these cases have been open safety pins. The difficulty encountered in the removal of these foreign bodies was due not to the mechanical problem of removal of the pin but to the obstruction to breathing due to pressure of the esophagoscope on the trachea. In order to make an esophagoscope with the smallest lumen practicable for work I had an esophagoscope made, using a 4 mm. inside lumen tube with a (one-piece) lamp-light carrier

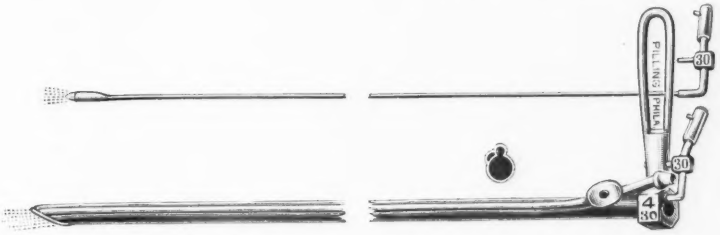


Fig. 1. One-piece light carrier lamp. Full lumen esophagoscope with outside diameter of 5 by 7 mm., circumference equal to 20 French bougie. Inside diameter 4 mm.

which I use in the infant bronchoscope A drainage canal was placed alongside the light carrier trough, as in the full lumen esophagoscope, with a thumb valve for the release of negative pressure. The instrument presents a full lumen of 4 mm. in diameter and a small outside diameter of 5 by 7 mm., equal in circumference to a No. 20 Fr. bougie, 30 cm. in length. (Fig. 1.) The infant esophagoscope and gastroscope is suggested for use in small infants for diagnostic esophagoscopy, for exploration of the lower esophagus or cardiac end of the stomach, for removal of safety pins from the esophagus by the intragastric rotation method, and for gastroscopic examination and removal of foreign body.

A FORCEP FOR MEASURING THE LARYNX.

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

In the direct laryngoscopic study of the infant larynx it was considered desirable to attempt various measurements during the examination. Several types of instruments were tried, including graduated bougies and a scaled rule with a handle at a right angle. These instruments were not practicable for taking all the measurements desired. With the direct laryngoscope in place it

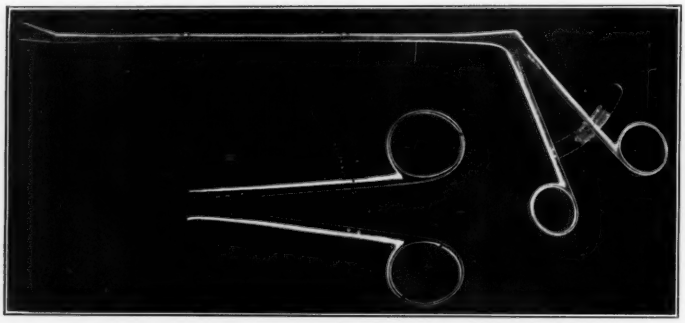


Fig. 1. Laryngeal measuring forcep, the Mathieu type, with a scale on the handle for determining the various laryngeal measurements. A thumb screw on the back of the instrument allows the forcep to be set at the measurement determined so that an accurate reading can be obtained.

was found that the jaws of the ordinary laryngeal forceps, known variously as the Mosher, Mathieu or Jackson forceps, when opened would span the longest diameter of the infant larynx. This style forcep was adapted for measuring the larynx, by attaching a scale to the handle indicating the width of opening the jaws in millimeters, and by cutting the tip of the forcep down until the closed jaws measured two millimeters. Starting with two millimeters, the scale was so adjusted that on opening the jaws a width of each millimeter could be read on the scale of the handle of the instrument. (Fig. 1.)

In the use of the instrument it has the advantage of being of the laryngeal forcep type with which we are all familiar. We have found it of value in the study of the infant larynx in both the

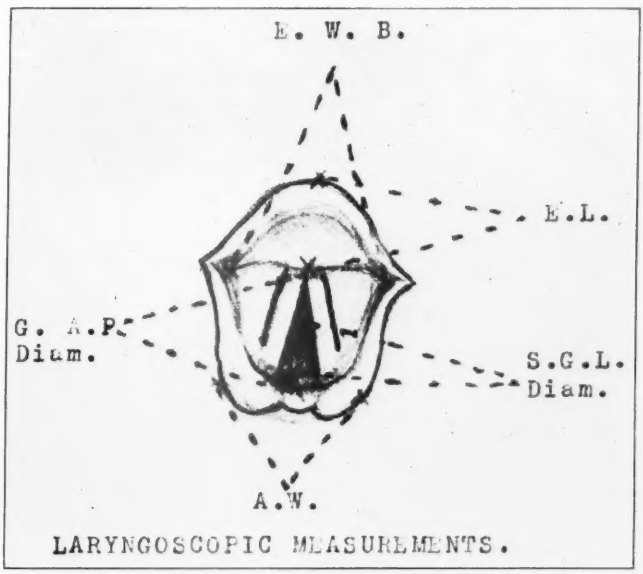


Fig. 2. A schematic drawing of the larynx with letters indicating the diameters easily obtained by use of the direct laryngoscopic measuring forceps. *E. W. B.*, epiglottis width at base; *E. L.*, epiglottis length; *G. A. P.*, glottis anteroposterior diameter; *S. G. L.*, subglottic larynx diameter; *A. W.*, arytenoid width. The length of the ventricle can also be readily measured with the forcep as well as the posterior glottic width on inspiration.

cadaver and living subject. The jaws of the forcep open widely enough to measure the subglottic larynx in the adult, the span of the instrument measuring 22 mm.

Various measurements were determined, width, length and breadth of the epiglottis, the anteroposterior diameter of the glottis, the length of the ventricle of the larynx, the width over the arytenoids and the subglottic diameter. (Fig. 2.)

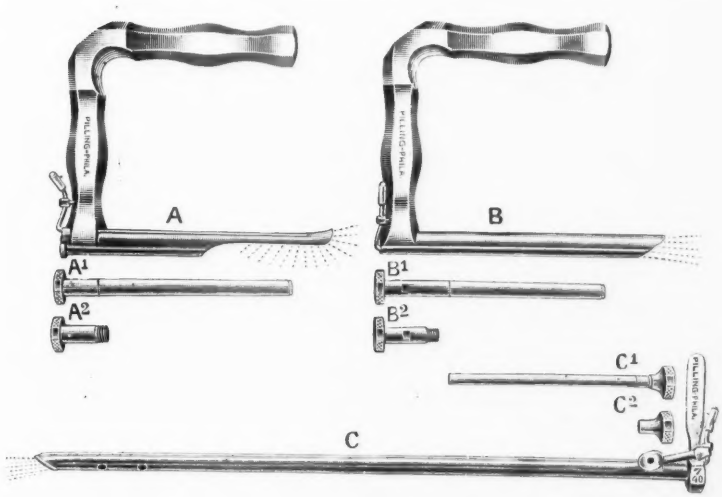
The laryngeal diameter that is of greatest practical value is the subglottic, because it is the smallest diameter and in the normal larynx cannot be enlarged without trauma, being surrounded by the cricoid cartilage. The subglottic diameter is also usually the dimension with which we are concerned in the passing of a bronchoscope. Disease conditions frequently involve the subglottic larynx, and this diameter then becomes the main consideration in laryngeal obstruction, either acute or chronic. The instrument will permit, also, the reading of the tracheal diameter some distance below the larynx. The use of the laryngeal measuring forcep is suggested for determining any measurements of the larynx through the direct laryngoscope, either in the infant child or adult.

THE LARYNGEAL MICROSCOPE, AND BRONCHOSCOPIC AND ESOPHAGOSCOPIC TELESCOPE.

GEORGE G. CARROLL, M. D.,

ROCHESTER, N. Y.

I wish to present to this Society a new instrument. It is essentially a compound microscope that fits into the direct laryngoscope. One of these (Fig. B-1) we have made small enough to fit the anterior commissure laryngoscope of the Jackson type (Fig. B); the other (Fig. A-1) fits the adolescent laryngoscope (Fig. A), Jackson type, or any larger caliber laryngoscopes of the same length. These microscopes when in place have a focus of about 3 cm. beyond the end of the laryngoscope. Of course, these microscopes can be fitted to any other type of laryngoscope on the market, but will have to be built according to the length of the laryngoscope. A microscope can also be made to fit an endoscope for



any of the other cavities of the body. These microscopes are low power, each having two eye pieces (Figs. A-1 and A-2; B-1 and B-2), one giving a magnification of 3 diameters and the other 6 diameters. The small microscope has three triplet lenses and one single. This gives it a stepping-up process of the image to be magnified by the eye piece. At the distal end is a disc of plain glass strong enough to be heated over an alcohol lamp to keep it from fogging. It is better to heat over an alcohol lamp rather than dipping the scope in hot water, as the glass at the end is not snugly fitted, as there must be a certain amount of play for contraction and expansion, otherwise there would be danger of fracturing the glass. The next instrument is virtually a telescope (Fig. C-1) of the Galileo type of the two lens system. It has a disc of plain glass suitable for heating over an alcohol lamp to prevent fogging. This telescope has two eye pieces (Figs. C-1 and C-2), one giving a magnification of 3 diameters and the other one of 4 diameters. This telescope can be used in the 7 mm. full lumen bronchoscope or esophagoscope of the Jackson type. In the esophagoscope, which is 45 cm. long, it has to be inserted almost up to the eye piece, but in the bronchoscope, which is usually 40 cm. long, it must be withdrawn about 5 cm. to get a focus. These

telescopes can also be made to fit any other endoscope, depending upon its length.

One would naturally raise the question in the case of the bronchoscope how the patient can breathe while this telescope is in position. We have found in all cases of diseased lungs, from young adults up, that there is sufficient leakage between the bronchoscope and the larynx, as well as air that passes down through the suction tube attached to the side of the bronchoscope, to give them enough air for respiration. In the severer type of lung infection it seems to be only necessary to flood the mouth with oxygen to quiet down the dyspnea. Oxygen can also be supplied through the

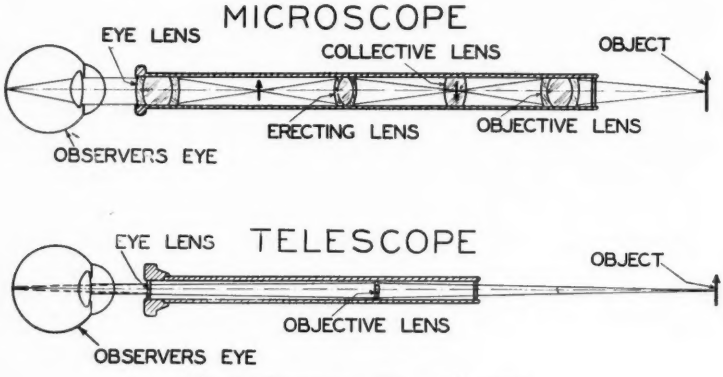


Fig. 1, Microscope. Fig. 2, Telescope.

suction tube at the side of the bronchoscope if necessary, using a small rubber bag as a guide to the amount of pressure to be used. We have not yet, after several months' experience in two clinics, had to resort to the small tube to supply the lungs with oxygen. In considering the small amount of air necessary to aerate the lungs one only has to think of the small caliber of an intubation tube when it is in position in the larynx and trachea.

Both the endoscopic microscope (Fig. 1) and telescope (Fig. 2) have been of great advantage to us in diagnosis, and a strong feature of these optical systems is that they in no way interfere with the routine work of endoscopy.

TWELVE-INCH SPECIMEN OF CARTILAGINOUS ESOPHAGUS.

HARRIS P. MOSHER, M. D.,

BOSTON.

Had this specimen come to me earlier, I should have asked for a place on the program, but I have had the specimen only a few days.

I have been finding a connection between infection of the gall bladder and of the liver and fibrosis of the lower end of the esophagus. The specimen I am showing is from a patient who died with atrophic cirrhosis of the liver. On account of the interference with the drainage of the esophageal mucosa in such a case, we would naturally expect dilated glands and an associated chronic infection. The pathological condition in this specimen, however, is new to me as well as to our pathologist.

The specimen was twelve inches long. As I took it in my hand, I noticed that the lower ten inches were firm and almost like cartilage. The upper two inches, however, were soft. On opening the esophagus, the musculature was found to be only slightly thickened. The lower end of the esophagus presented a firm, whitish, marble-like tumor, 1½ cm. in diameter, and one or two smaller tumors of the same nature. It was found that the whole of the esophagus, except the first two inches, was studded with dilated glands. The firm feeling of the esophagus was apparently due to these dilated glands and the jelly-like material contained in them.

The pathologic condition which caused these cysts was found to be a proliferation of the surface epithelium downward along the gland ducts and the plugging of the ducts by the proliferated epithelium. As I said, our pathologist has never seen this condition in the esophagus but he has seen an analogous condition in cirrhosis of the breast. The question in this case is whether the change in the epithelium is a beginning form of carcinoma. I never knew carcinoma of the esophagus to start over an area as extensive as ten inches. Another possible cause for this condition is some form of vitamin deficiency. Certain animals, whose diet is mostly cereal, have a keratosis of the esophageal epithelium normally.

IMPROVED HYDROSTATIC DILATOR FOR USE IN CARDIOSPASM.

CHARLES J. IMPERATORI, M. D.,

NEW YORK.

This instrument that I am presenting before the Society is an improved Plummer, Mosher hydrostatic dilator. The original Plummer dilator consisted of a rubber tube to which was attached a rubber bag surrounded by a silk bag and this enclosed in an outer rubber bag. At the end of the instrument a steel olive was attached. In order to overcome the flexibility of the instrument a whalebone bougie was permanently left in the inner portion of the tube. This part of the instrument was attached to the water faucet by another tube and in turn connected by a "Y" to a manometer. A certain marking on the tube indicated when the dilating portion of the instrument was in the cardia. Mosher suggested the use of a radiopaque mixture and the placement of the instrument in the cardia with the assistance of the fluoroscope. In Mosher's later instruments, instead of using the fluid dilating media, air was used and the bag was painted with strips of barium. In order to avoid possible rupture of the bag, either through air pressure, as in the Mosher instrument, or water pressure from the faucet, as in the Plummer instrument, the instrument (Fig. 1) presented for your consideration is actuated by a syringe with a two-way valve so that fluid may be pumped into the bag and by the use of a small inner tube the air that is in the outer portion of the tube may be ejected and nothing but water contained in the dilating bag. The instrument is introduced into the patient while under the fluoroscope and guided into the cardia by this means.

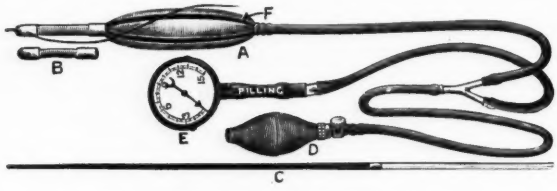
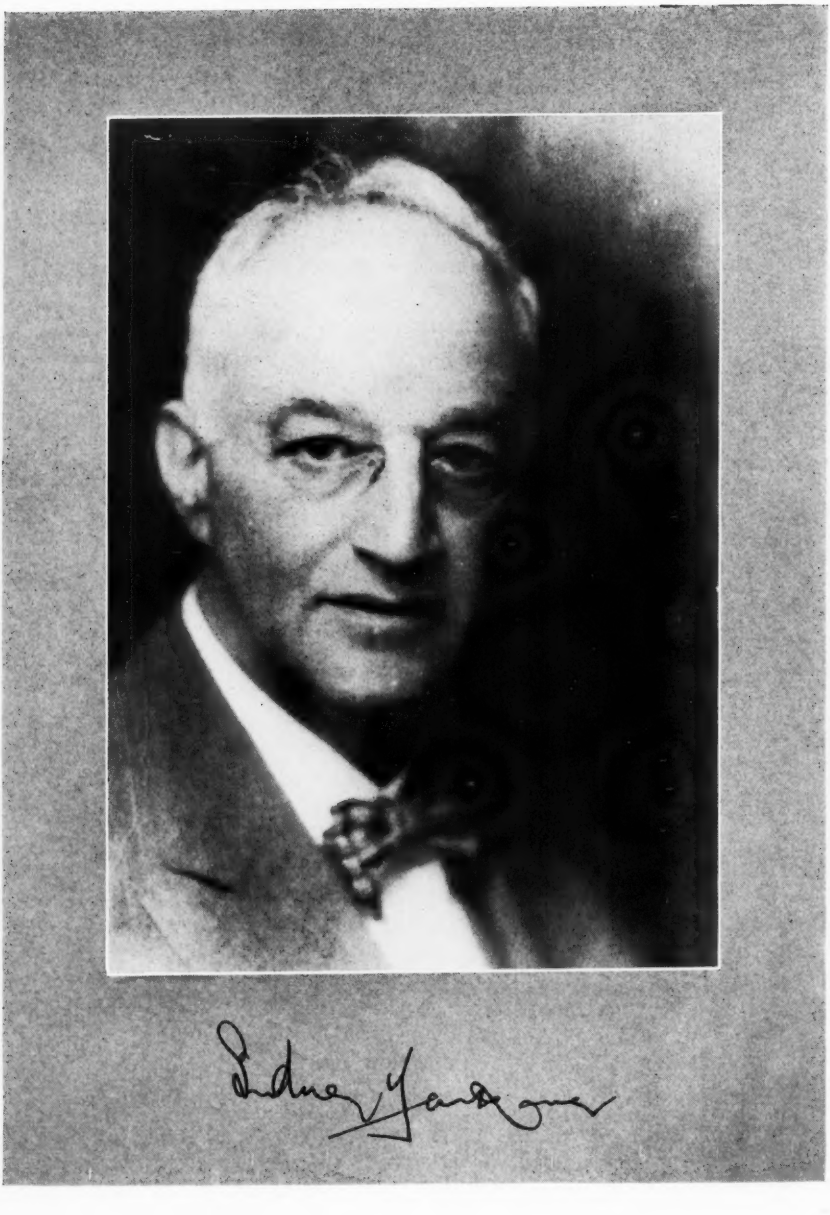


Fig. 1. Imperatori Esophageal Dilator.

Dilatation is accomplished by pumping the fluid into the dilating bag, noting the pressure on the manometer and observing the dilatation under fluoroscopic guidance. The reason for attempting to have entirely hydrostatic dilatation is that where air is put under pressure, if a rupture of the bag were to occur, more serious results would follow than if the bag merely ruptured by the use of water. In the Plummer instrument there was always a mixture of air and water. The success obtained in hydrostatic dilatation of the cardia is in sustaining the dilatation in from eight to ten minutes' time. This is not a completed instrument. Theoretically it should have worked perfectly, but there has been constantly the danger of the small inner tube kinking and the air not being released. Further work must be given to this, and it is hoped at some subsequent meeting a perfected instrument can be demonstrated. The successful treatment of cardiospasm was added to materially by Mosher's suggestion in the use of a barium mixture instead of water and the guidance of the dilator into the cardia and retaining of it in proper position by the use of the fluoroscope. I am certain that considerable success of this method of treatment has been added to by the sustaining of the dilatation over an appreciable period of time—say ten to twelve minutes.



SIDNEY YANKAUER.

Dr. Sidney Yankauer died August 26th of heart disease at Mount Sinai Hospital after a brief illness. He was 60 years old.

Born in New York City, Dr. Yankauer was graduated with an A. B. degree from the City College in 1890, and received his M. D. degree from Columbia University's College of Physicians and Surgeons three years later. After an internship at Mount Sinai Hospital, he began practice in 1893.

At his death Dr. Yankauer was laryngologist at Mount Sinai and consulting laryngologist at Beth Moses Hospital and at St. Joseph's, Far Rockaway. He was also bronchoscopist at Montefiore, Broad Street, Beth David and Beth Israel Hospitals.

During the World War he served overseas as a Major in the Army Medical Corps.

He belonged to the New York Academy of Medicine, the New York County Medical Society, the American Laryngological, Rhinological and Otological Society, the American Bronchoscopic Society, the American Academy of Ophthalmology and Otolaryngology, the State Medical Association, and the American Thoracic Surgical Society. He was a Fellow of the American College of Surgeons.

Dr. Yankauer was a mechanical genius and the originator of many surgical instruments now in common use. Among them are the pharyngeal speculum for direct examination of the eustachian orifice; an electrode for fulgurating the larynx; a radium needle for the esophagoscope and many endoscopic instruments.

Surviving are a widow, Mrs. Margaret K. Yankauer; a daughter, Miss Alice Yankauer; a brother, Theodore, and three sisters, Mrs. Ida Heilbrun, Mrs. Martha Jones and Mrs. Lena Dublin, all of New York.

Dr. Yankauer wrote much for medical publications. A list of his writings follows:

THE PUBLICATIONS OF DR. SIDNEY YANKAUER.

An Unusual Case of Empyema of the Antrum of Highmore. Medical Record, August 15, 1903.

Tuberculosis of the Larynx. The Laryngoscope, March, 1904.

Sounds for the Eustachian Tube. The Laryngoscope, May, 1906.

A Laryngeal Medicator for the Patient's Use. The Laryngoscope, August, 1907.

A Case of Carcinoma of the Nasopharynx and Sphenoid Region Which Responded Favorably to the Use of Radium. The Laryngoscope, May, 1908.

The Drainage Mechanism of the Normal Accessory Sinuses. The Laryngoscope, July, 1908.

Tonsillectomy. The Laryngoscope, May, 1908.

The Incision for the Submucous Resection. Annals of Otology, Rhinology and Laryngology, December, 1909.

The Isthmus of the Eustachian Tube: A Contribution to the Pathology and Treatment of Middle Ear Diseases. The Laryngoscope, July, 1910.

Foreign Body Removed from the Bronchus. The Laryngoscope, November, 1910.

The Pharyngeal Orifice of the Eustachian Tube With a Description of a Speculum and Other Instruments for the Direct Examination and Treatment Thereof. Read before the American Laryngological, Rhinological and Otological Society at Atlantic City, June, 1911.

Four Cases of Foreign Body in the Esophagus Removed With the Aid of the Esophagoscope. Two Cases of Foreign Body in the Bronchus; A Case of Tumor in the Lower Trachea: Removed by Upper Bronchoscopy. Annals of Otology, Rhinology and Laryngology, June, 1911.

The Salpingian Curette. The Laryngoscope, May, 1912.

Three Cases of Foreign Body in the Bronchus. The Laryngoscope, October, 1912.

The Technic of Intranasal Operations Upon the Lacrimal Apparatus. The Laryngoscope, December, 1912.

The Conservative Treatment of Chronic Aural Suppuration. Annals of Otology, Rhinology and Laryngology, December, 1912.

An Electrode for Fulgurating the Larynx. The Laryngoscope, December, 1914.

Report of a Collective Investigation on the Curettement of the Eustachian Tube in Chronic Aural Suppuration. The Laryngoscope, October, 1915.

Bronchiectasis From the Standpoint of the Bronchoscopist. New York Medical Journal, February 5, 1916.

Experiences in Esophagoscopy. American Journal of Surgery, March, 1917.

The Fossa of Rosenmüller from the Rhinological Standpoint. The Laryngoscope, December, 1917.

Symposium on Borderline Diseases. The Laryngoscope, February, 1921.

The Complete Spheno-Ethmoid Operation. The Laryngoscope, November, 1921.

Two Cases of Lung Tumor Treated Bronchoscopically. New York Medical Journal and Medical Record, June, 1922.

A Radium Needle for the Esophagoscope. Archives of Surgery, January, 1923.

Eustachian Tubal Curettage. The Laryngoscope, March, 1925.

A New Tonsil Irrigator. The Laryngoscope, March, 1925.

A New Laryngeal Syringe. The Laryngoscope, October, 1927.

Bronchoscopy in Diseases of the Lung. Presidential Address Delivered at the eleventh annual meeting of the American Bronchoscopic Society at Washington, D. C., April 30, 1928.

Cyst of the Lung. Read before American Bronchoscopic Society, May, 1930.

Nasopharyngeal Abscess; A Report of One Hundred and Fifty Cases. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, June, 1930.

Instruments for the Dilation of the Pharyngeal Orifice of the Eustachian Tube. The Laryngoscope, February, 1932.

NOTICES.

The first mid-winter course in Ophthalmology and the second mid-winter course in Otolaryngology will be held in Los Angeles from January 16 to January 27, 1933, inclusive. Later announcement of the personnel of the teaching staff will be made.

American Laryngological, Rhinological and Otological Society

The next annual meeting of the American Laryngological, Rhinological and Otological Society will be held in Chicago, Ill., June 8, 9 and 10, 1933, under the presidency of Dr. Joseph C. Beck. This meeting will follow the opening of the "Century of Progress" Fair in Chicago and will immediately precede the meeting of the American Medical Association in Milwaukee.

Robert L. Loughran, Secretary.

The American Board of Otolaryngology.

An examination was held in Montreal, Canada, September 17, 1932, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Seventy-three candidates were examined, of which sixteen were conditioned or failed.

The Board will hold an examination in Milwaukee, Wisconsin, June 12, 1933, during the meeting of the American Medical Association. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for proper application blanks.

W. P. Wherry, M. D., Secretary-Treasurer.

H. P. Mosher, M. D., President.

Abstracts of Current Articles.

NOSE.

Treatment of Nosebleed (Traitement des saignementa de la cloison nasale).

H. Lewenfisz (Warsaw), Ann. Otolar. 2:295, March, 1932.

Detachment of the bleeding mucosa by injection of a few cubic centimeters of one-half percent novocain is the essential of this method. If necessary, anterior incision, with elevation of mucosa from the perichondrium is made and packing inserted and left twenty-four hours. FENTON.

Saline-iodide Inhalations: Effects on the Leukocytic Formula in Adenoidectomized Children (Terapia inalatoria salsoiodica e variazioni della formula leucocitaria ecc.).

Dr. M. Mancioli (Rome), Valsalva, 8:210, March, 1932.

Sixty children, whose adenoids had been removed at least three months before, had differential white counts and were then exposed for thirty minutes daily during two weeks to the hot saline-iodin vapors of the natural springs of Salsomaggiore, in the Institute for the Cure of Adenoidism. Marked increase in neutrophils and decrease in lymphocytes took place in all cases, along with considerable general physical improvement. Open nasal passages are necessary for this kind of treatment to be of any avail. FENTON.

Presentation of Instruments and Specimens.

Dr. Thomas E. Carmody (Denver), Trans. Am. Laryngol. Assn., May, 1932.

Osteomas in the sinuses are rare.

A man, aged 26, a medical student, had been seen by a number of nose and throat men and once by our President, Dr. Shambaugh, with a severe headache. The headache was relieved after two or three days as a rule, because drainage occurred from the sinus, but roentgenograms were not taken until after he had had severe headaches for almost four years, and he had had them almost continuously for the last four months. The osteomas were both in the left frontal sinus. They interfered with the drainage sufficiently so that the symptoms were produced.

In this case an ordinary Lynch operation was done and the specimen removed, and entire freedom was obtained. However,

there is one peculiar thing about this case. In the opposite sinus, the right frontal, there was an apparent polyp. The polyp in the other sinus remained, and there was a possibility that there was a potential osteoma in that sinus.

The second patient was a man, aged 37, a war veteran, who gave a history of having blown his nose three years previously, with emphysema of both of the eyelids on the right side. There was apparently no examination at that time. On January 1, 1932, he developed an aphasia while talking business, and was unable to proceed with his business arrangements, but he recovered his voice on returning home that evening. The next morning he found that he was paralyzed on the right side, the arm and leg; the paralysis remained for some days, when he began to recover the use of the right arm. The leg had not recovered or had recovered very little at the time I saw him, on April 14th.

A Killian operation was performed on April 17th, and an attempt to do the ordinary operation, like the Lynch, resulted in inability to remove the specimen. The specimen was attached to the posterior wall of the frontal sinus, and when it was pulled away there was a hissing sound. Apparently quite an amount of air or gas and some fluid was present. There was an opening through the dura, the dura was necrotic, and there was apparently an abscess of the brain. The lateral roentgenogram showed the air extending back over the parietal region. Recovery was uneventful, and the patient was recovering somewhat the use of his leg. After leaving the hospital he was placed under my care in the Veterans' Bureau Hospital. I saw him on the day I left Denver and he was in good physical condition. IMPERATORI.

The Anatomic Relation of the Sphenoid Sinuses to Dorello's Canal: Abducens Paralysis.

Dr. Karl Musser Houser (Philadelphia), Trans. Am. Laryngol. Assn., 1932.

In thirty-eight sphenoid cases measurements were made of the distance between Dorello's canal and the nearest part of the interior of the sinus. Ten and five-tenths per cent showed a distance of 2 mm. or less. The idea is advanced that in certain cases of sphenoidal inflammation, paralysis of the sixth nerve may be secondary to an inflammatory reaction in the region of Dorello's canal. IMPERATORI.

PHARYNX

Chancre of Lingual Tonsil (Sifiloma iniziale della tonsilla linguale ecc.).

Dr. G. Scalori (Pisa), Valsalva, 8:372, May, 1932.

Here is a careful differential study of an ulcerating initial lesion of two months' standing, far down at the base of the tongue, extending into the lower part of one faucial tonsil. Diagnosis was based on positive blood Wassermann, characteristic plasma cell infiltration of biopsy specimen and immediate cure under antiluetic injections. Ulceration had occurred because of secondary infection of the lesion by Vincent's organisms. FENTON.

LARYNX.

Chondroma of the Larynx: A Report of Six Cases.

Dr. Frederick A. Figi (Rochester, Minn.), Trans. Am. Laryngol. Assn., May, 1932.

Knowledge concerning the clinical characteristics of chondroma of the larynx, permitting early recognition and effective treatment, accumulates slowly because of the rarity of these tumors. A review of the symptoms, diagnostic features and treatment, together with case reports of six chondromas of the larynx seen in the Mayo clinic, are presented. IMPERATORI.

The Treatment of Cicatricial Stenosis of the Larynx, With Special Reference to the Application of Surgical Diathermy.

Dr. M. F. Arbuckle (St. Louis), Trans. Am. Laryngol. Assn., May, 1932.

A brief consideration of the methods now employed in the treatment of cicatricial stenosis was presented, with a review of the recent literature on the subject and a further report on the use of skin graft to prevent the recurrence of scar tissue in the larynx.

Dr. Arbuckle emphasized the necessity for careful and repeated observation of the larynx during quiet respiration in order to gain more accurate information of the distribution of scar tissue and of the possible interference with or departure from normal motility of the larynx.

The plan followed in applying surgical diathermy for the removal of scar tissue from the larynx was described, with a comparison of the results obtained by this and other procedures.

Anesthesia is, of course, necessary; chloroform is used when diathermy is to be employed.

Notes regarding special preoperative preparation of the patient and postoperative care, including indications of damage by chloroform, were discussed.

The use of carbon dioxide and oxygen in all postoperative cases as a means of prevention of pulmonary complications was discussed. IMPERATORI.

Laryngitis and Tracheitis in Children, With Special Reference to Nondiphtheritic Infections.

Dr. T. R. Gittens (Sioux City, Iowa), Trans. Am. Laryngol. Assn., May, 1932.

Nondiphtheritic laryngitis and tracheobronchitis simulates true laryngeal diphtheria and is often confused with it. In severe cases, tracheotomy is necessary to relieve serious dyspnea, just as in diphtheria. In the nondiphtheritic type there is a marked tendency for secretions and semisolid masses to form in the trachea and bronchi and produce mechanical obstruction of a serious nature for days or even weeks after tracheotomy. Suction through the cannula, together with instillation of weak epinephrine or saline solution to liquefy secretions, is of great importance in the relief of post-tracheotomy dyspnea. The giving of fluids freely by mouth, rectum or intravenously is the most important part of the general treatment. Tracheotomy is preferred rather than intubation, for various reasons given in the paper.

Twenty-four personal cases of nondiphtheritic laryngitis and tracheobronchitis were discussed. IMPERATORI.

MISCELLANEOUS.

Valsalva's De Aure humana.

Italian translation by Prof. V. Manedno, introduction by Prof. G. Bilancioni; ten reproductions of the original plates. pp. 164, Rome, Il Valsalva, Sept., 1931.

This is a handsome reprint of an epochal work, with erudite and spirited comments by Ferreri's successor in the Roman Policlinic. The original plates are splendidly reproduced in all their remarkable accuracy and brilliance. FENTON.

Relative Sizes of Nasopharynx, Accessory Sinuses and Mastoids (Rapporti tra hampiezza delle cavita rinofaringee deiseni, dede mastoide).
Prof. G. Bilancioni (Romè), Arch. Radiol., 10:480, May, 1932.

Radiologic comparison of sinus and petromastoid development discloses three types: 1. No differentiation of the petromastoid cell complex, slight facial pneumatization; 2. Marked mastoid pneumatization with normal facial cell structures; 3. Diploetic petrous and mastoid with very roomy multilocular accessory sinuses.

FENTON.

Physical Therapy in Otorhinology (La terapiafisica nel traftamento delle mallattie del naso-gola-orecchio).
Prof. F. Lasagna (Parma), Boll. Malatt. Orecch. Gola, Naso, 50:176, June, 1932.

The author recommends surgical diathermy for discrete forms of rhinophyma, rhinoscleroma, turbinal hyperplasias, benign tumors, rhinopharyngeal fibromas and malignancies; for tonsil work in hemophilics and the aged, and both procedures in benign and malignant laryngeal tumors, and for scleroma and infiltrative tuberculomata; associated with the surgical approach, for control of benign and malignant aural neoplasms; for reduction or destruction of tumors and cicatricial stenoses of the esophagus, by means of appropriate endoscopy. Ordinary forms of local anesthesia, with atropin to reduce secretion, are employed.

Medical diathermy, avoiding great elevation of local temperatures, is valuable in otosclerosis and exudative deafness with losses of high and low tones, not in purulent processes.

It is useful in sinusitis only to reduce the pain of acute and subacute frontal sinusitis, and good drainage must be present to forestall the constant danger from diathermic congestion. Furunculosis of the ear and simple acute rhinitis are benefited by a few cautious applications.

Ultraviolet radiation is worthless in external otitis; it may help in chronic suppuration where a large perforation exists. Acute rhinitis is helped by joint use of the quartz lamp and colloidal silver; also lupus of the skin and mucosa. Chronic rhinitis and sinusitis are not benefited. Lasagna recommends the quartz lamp for Vincent's angina, but as he simultaneously uses acriviolet and chromic acid, the value of irradiation seems doubtful.

Laryngeal tuberculosis is greatly helped, not only by direct sunlight and the laryngeal mirror, but also by the small Kromayer lamp through the direct laryngoscope. This does not replace local use of trichloracetic acid for ulcerations and galvanocautery for infiltrations.

The galvanic current used with electrolytes is only helpful in suppurative otitis where broad perforations exist. Its main value is as a caustic for removal of papillomata, and destruction of angiomas and varicosities. He also suggests its use for the pain of ophthalmic or aural herpes zoster.

Infra-red rays are helpful for acute frontal sinus pain.

Lasagna denies any value to recent electric instruments which transmit musical tones, for the relief of any form of deafness.

FENTON.

Temperature Control by Special Vasculo-sensory Apparatus in the Organs of Touch, Taste and Smell (Dispositifs vaso-sensorièls Spécialises au niveau des organes du tact, de la gustation et de l'olfaction. Leur rôle de thermostabilisation).

Dr. M. H. Valette (Bordeaux), Rev. de Lar., Otol. and Rhinol., 53:553, May, 1932.

Following Prof. Portmann's new editorial principle, this important thesis of forty pages details the minute anatomy of numerous organs, hair, taste buds and olfactory turbinals, pointing out the necessity for sympathetic control of the vascular plexuses in these organs for protection of their delicate nerve terminals, while deeply situated sensory organs like the ear do not require such mechanisms.

FENTON.

Action of Tonsil Extract on Ovarian Function (Azione dell'estratto tonsillare con speciale riguardo alla funzione ovarica).

Dr. G. DeMaria (Rome), Valsalva, 8:321, May, 1932.

Daily injections of fresh extract of tonsils from young individuals were made during many months into female white rats. This resulted in a marked shortening of menstruation and delay in its appearance. Marked hyperplasia of all lymphatic tissues (lymph glands, spleen, thymus) and follicular atresia and diminution of functionating corpora lutea in the ovaries were notable pathologic findings. He therefore ascribes to the tonsil an endocrine function on the ovary and entire organism similar to that of the thymus.

FENTON.

The Treatment of Allergy Based on the Conception That It Is a Potential Alkalosis.

Dr. Arthur M. Alden (St. Louis), Trans. Am. Laryngol. Assn., May, 1932.

The results obtained from protein desensitization therapy in allergic cases have been disappointing. An explanation was offered of the conception that an alkalotic background, congenital or acquired, is an important factor in allergy. The rationale of the therapy is based on such a conception. Cases were reported.

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